"Efficacy of Abdominal risk model in predicting the probability of wound dehiscence"

Dissertation submitted to

The Tamil Nadu M.G.R Medical University

Chennai- 600032, April-2014

In partial fulfillment of the

Regulations of the award of degree of

M.S. General Surgery

Department of General Surgery

Coimbatore Medical College Hospital

Coimbatore - 641018

CERTIFICATE

This is to certify that this dissertation titled ***"Efficacy of abdominal risk model in predicting the probability of wound dehiscence"*** submitted to the Tamil Nadu Dr. M.G.R. Medical University, Chennai in partial fulfillment of the requirement for the award of M.S Degree Branch - I (General Surgery) is a bonafide work done by **Dr.Sudish D**., post graduate student in General Surgery under my direct supervision and guidance during the period of September 2012 to November 2013.

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DECLARATION

I hereby declare that the dissertation entitled ***"Efficacy of abdominal risk model in predicted probability of wound dehiscence"*** was done by me at Coimbatore Medical College Hospital Coimbatore – 641018 during the period of my post graduate study for M.S. Degree Branch-1 (General Surgery) from 2011 to 2014.

This dissertation is submitted to the Tamil Nadu Dr. M.G.R. Medical University in partial fulfillment of the University regulations for award of M.S., Degree in General Surgery.

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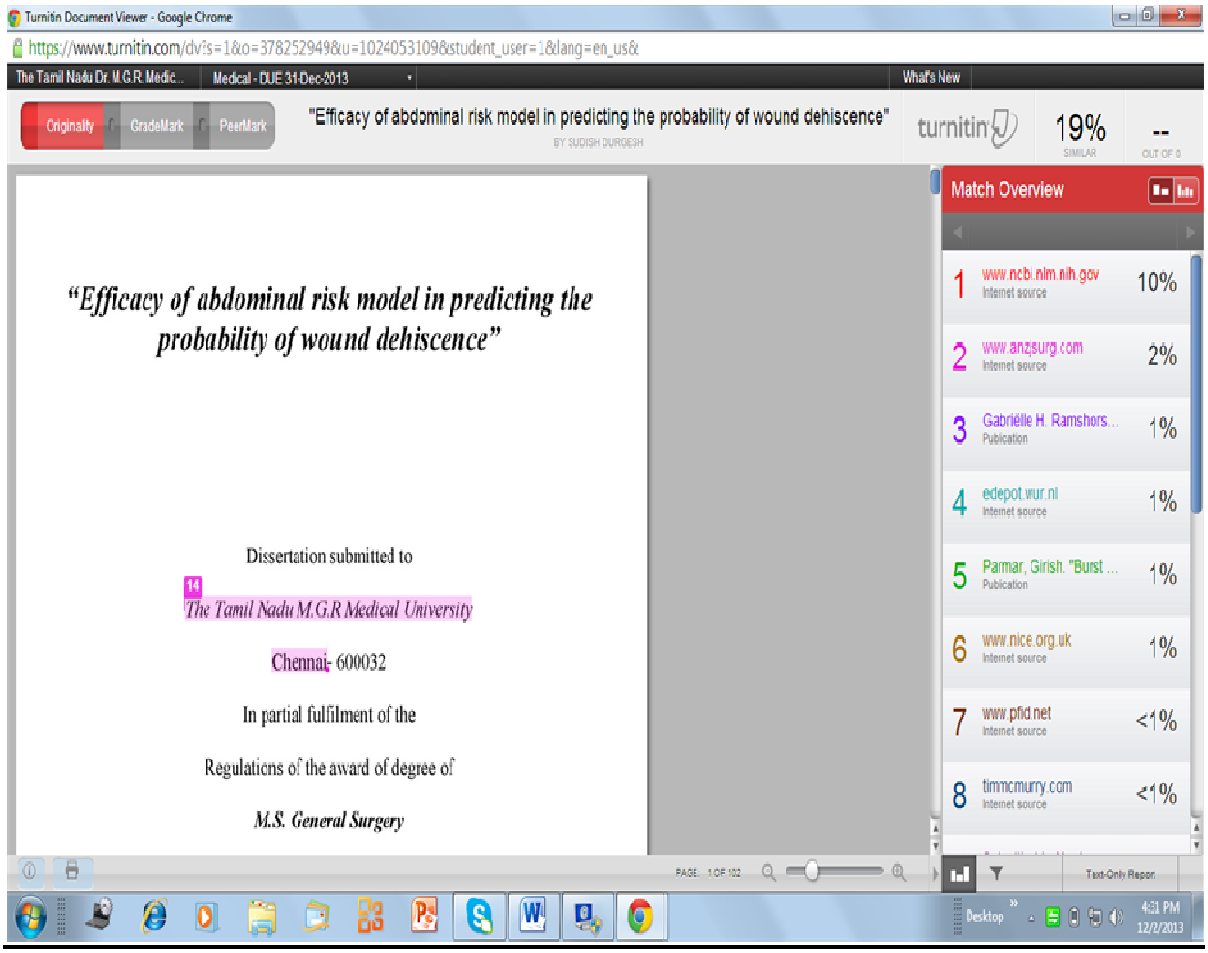


“EFFICACY OF ABDOMINAL RISK MODEL IN PREDICTING THE PROBABILITY OF WOUND DEHISCENCE”

TABLE OF CONTENTS

ABSTRACT

BACKROUND AND OBJECTIVES

Wound dehiscence or burst abdomen is a grave post-operative complication associated with a high morbidity and suffering to the patient. It has significant impact on health care cost, both for the patient and the hospitals.

Several studies have been performed to identify risk factors for abdominal wound dehiscence. Very few risk models have been developed to predict the probability of occurrence of dehiscence. One such scoring system is the abdominal risk model developed by Department of General surgery, Erasmus university medical center, Netherlands which is based on the relative weights of various risk factors. The model was validated in a separate population and demonstrated high predictive value for abdominal wound dehiscence.

The **need for this study** is to highlight the efficacy of the scoring system to predict the probability of wound dehiscence and stratify the risk accordingly so that in future timely interventional strategies can be instituted thus preventing the occurrence of burst abdomen.

The association and prevalence of various risk factors in occurrence of the dehiscence were identified in the study. The incidence rates associated with emergency surgeries, pattern of occurrence and management of wound dehiscence were also studied.

METHODS

A total of 100 cases undergoing midline laparotomies were included in the study. Each case examined clinically and properly in systematic manner and an elaborative study of history based on chief complaints, significant risk factors, investigations, time and type of surgery performed and postoperative events and day of onset of wound dehiscence. Calculation of probability of wound dehiscence (P) was calculated according to the logistic formula: $P = e^x/ (1+e^x) X 100\%$;where 'e^x' represents the exponential function and x represents '-8.37 + (1.085 X Calculated total risk score)'. The total risk score is calculated by adding the weights of the various risk variables. Then we compare this probability score with the actual outcome of the patient with regard to the occurrence of abdominal wound dehiscence in each risk score group.

RESULTS

A total of 13 patients developed wound dehiscence all occurring in males, belonging to the age group of 5^{th} to 6^{th} decade. Chronic obstructive pulmonary disease and anaemia were the major pre-operative comorbid factors associated with burst abdomen. . Post-operative abdominal wound dehiscence is more common in patients operated in emergency and in those in a setting of wound infection.

The predicted probability calculated by the abdominal risk model slightly underestimated the actual occurrence of wound dehiscence in each risk score group, although there was a noticeable exponential increase in occurrence of abdominal wound dehiscence with increase in risk score. By this we were able to stratify the patients into high risk and low risk group based on the risk score.

CONCLUSION

Hence with the help of this risk model we will be able to classify and triage the patients to risk groups in whom timely preventive strategies can be instituted and thus reducing the occurrence of dehiscence. Knowledge of the common risk factors, early diagnosis and effective management helps in reducing the morbidity and mortality of this complication.

KEYWORDS- abdominal wound dehiscence, burst abdomen, abdominal risk model, midline laparotomies, wound infection.

INTRODUCTION

Abdominal wound dehiscence (burst abdomen, fascial dehiscence) is a grave post-operative complication described as partial or complete disruption of an abdominal wound with or without protrusion and evisceration of abdominal contents.There are two basic types of wound dehiscence, partial or complete, depending on the extent of separation. In partial dehiscence, only the superficial layers or part of the tissue layers reopen. In complete wound dehiscence, all layers of the wound thickness are separated, revealing the underlying tissue and organs, which may protrude out of the separated wound.

The importance of this clinical entity is due to the fact of it causing significant impact on general population. The suffering comes from the fact that patient is subjected to inconvenience of a discharging wound and the later appearance often incisional hernia. It is one of the dreaded complication faced by surgeons and of greatest concern as it carries with it the risk of evisceration. It may need immediate intervention and there is a possibility of recurrence. Surgical wound infection can also occur with incisional hernia formation. Incidence as described in westernliterature is 0.4%-3.5% and in Indian literature is 1-2%[1,2,3]. Mortality and morbidity in the form of prolonged hospital stay, increased economic burden on health care resources and long term

complication of incisional hernia can be reduced by highlighting the risk factors for wound dehiscence.

Dehiscence in simple terms refers to mechanical failure of wound healing. Various factors that affect wound healing influence the outcome in occurrence of wound dehiscence. Therefore a good knowledge of the risk factors affecting wound healing is necessary in preventing its recurrence. Several risk factors identified which are responsible for wound dehiscence can be broadly classified into

- Patient related factors-advanced age>65yrs, systemic diseases(uremia, diabetes mellitus), malnutrition(hypoalbuminaemia ,anemia)
- Operative related factors include emergency surgery and type of surgical wound.
- Post-operative factors includes cough ,vomiting and distension.

Despite advances in perioperative care and suture materials, incidence and mortality rates in regard to abdominal wound dehiscence have not significantly changed over the past decades. This may be attributable to increasing incidences of risk factors within patient populations outweighing the benefits of technical achievements.

Several studies have been performed to identify risk factors for abdominal wound dehiscence. No risk model has yet been developed to predict the probability of wound dehiscence.In the study conducted by

Van Rams horst et al independent risk factors for abdominal wound dehiscence were identified and a risk model was developed to recognize patients with high risk for abdominal wound dehiscence.Major independent risk factors identified were age, gender, chronic pulmonary disease, ascites, jaundice, and anemia, and emergency surgery, type of surgery, postoperative coughing, and wound infection. Risk model designed was based on relative weights of various riskfactors. Resulting scores ranged from 0 to 8.5, and the risk for abdominal wound dehiscence over this range increased exponentially from 0.02% to 70.1%.The model was validated in a separate population and demonstrated high predictive value for abdominal wound dehiscence supporting the fact that variable identified as risk factors are actual risk factors. With the help of this model based on patients risk factors the probability of post-operative wound dehiscence can be calculated. Following are the advantages of scoring system in predicting the probability of wound dehiscence-.

- Helps in stratification and identifications of patients at risk of developing wound dehiscence.
- Role of peri-operative factors can be understood and mitigated in reducing the occurrence of dehiscence.

Timely intervention of high risk patients that involves preventing wound closing with suitable reinforcements, thus preventing the occurrence of dehiscence.

OBJECTIVES

1. To study the efficacy of scoring system in predicting the probability of wound dehiscence and to stratify the risk accordingly.
2. To assess the association and prevalence of risk factors in occurrence of abdominal wound dehiscence.
3. To study the incidence rates associated with elective and emergency surgery, pattern of occurrence and management of wound dehiscence.

REVIEW OF LITERATURE

ANATOMY OF ANTERIOR ABDOMINAL WALL[4,5,6]

Anteriorly, the abdominal wall extends from the surface of the xiphoid process (level of the ninth thoracic vertebra) to the pubic symphysis (level of coccyx). Laterally and posteriorly, the abdominal cavity is overlapped by the thorax superiorly and by the gluteal region of the lower limb inferiorly.

ANTEROLATERAL ABDOMINAL MUSCLES

The three muscle layers of the body wall are separate in the flanks, where they are known as external oblique, internal oblique and trasversus abdominis muscles. The layers fuse together ventrally to form the rectus sheath. On each side the muscles of this wall end inferiorly at the inguinal ligament though the fascia superficial to them extends to the fold of the groin before fusing with the deep fascia of the thigh (fascia lata).Superficial fascia of the anterior abdominal wall consists of superficial fatty (Camper's Fascia) and deep membranous layers (Scarpa's Fascia).

These are in three layers, each of which is muscular posterolaterally and aponeurotic anteromedially. The external and internal oblique muscles are approximately fan shaped.

EXTERNAL OBLIQUE

This muscle arises from eight digitations one from each of the lower eight ribs on their external surfaces and radiates downwards and forwards. The muscle fans out to a very wide insertion much of which is aponeurotic. The lower border lying between the anterior superior iliac spine and the pubic tubercle forms the inguinal ligament.

INTERNAL OBLIQUE

It arises from the lumbar fascia, the iliac crest, and the lateral two thirds of the inguinal ligament and radiates upwards and forwards. Below the costal margin, the aponeurosis splits around the rectus muscle. The posterior layer below the umbilicus ends in a curved free margin, concave downwards, the arcuate line or semi circular line of Douglas. The internal oblique has a free lower border which arches over the spermatic cord.

TRANSVERSUS ABDOMINUS

It is the innermost layer, is horizontally disposed. It runs from the internal surface of the rib cage, the lumbar fascia, the iliac crest and the lateral third of the inguinal ligament to the lineaalba. The muscle fibres become aponeurotic and pass behind the rectus to fuse with the internal oblique aponeurosis into the lineaalba. Its fibres lie at an angle to the intermediate fibres of both the other muscles but are parallel to

those of the external oblique superiorly and to the internal oblique inferiorly.

The lower parallel fibres of the aponeurosis of the internal oblique and transversusabdominis do not reach the lineaalba but fuse to form the conjoint tendon.

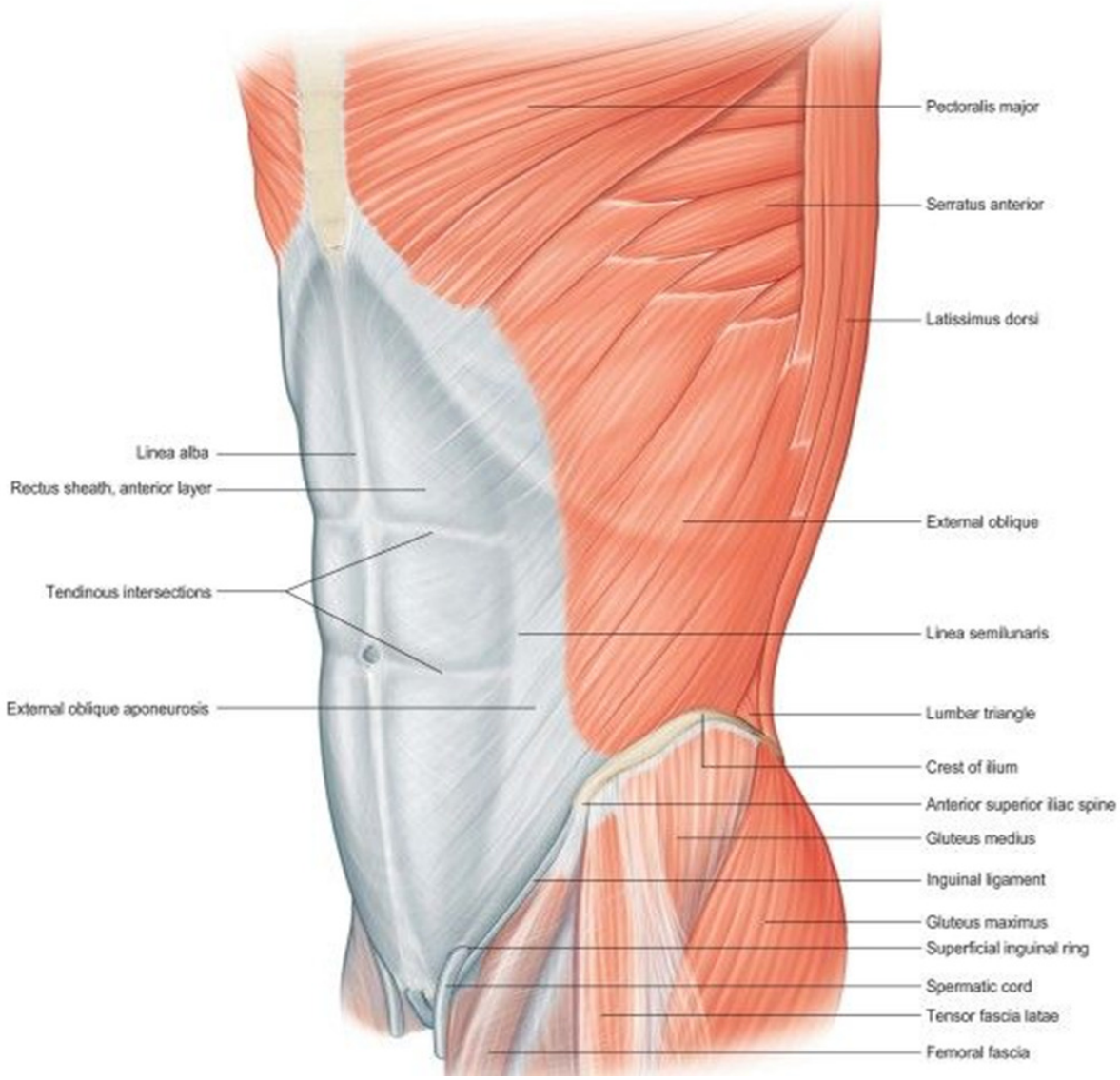


FIGURE -1: ANATOMY OF ANTERIOR ABDOMINAL WALL (5)

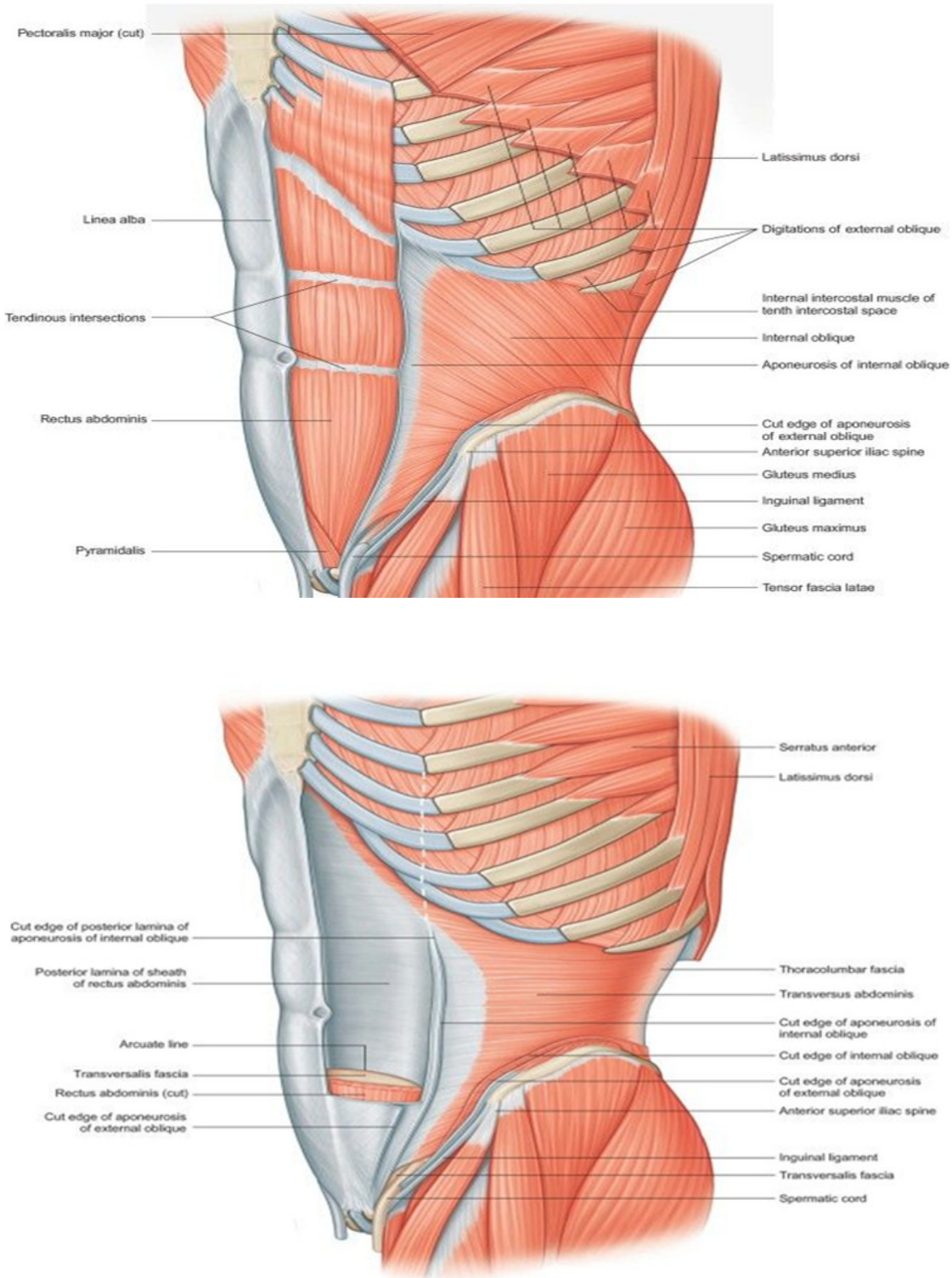


FIGURE-2: ANATOMY OF ANTERIOR ABDOMINAL WALL EXPOSING INTERNAL OBLIQUE AND TRANVERSUS ABDOMINIS(5)

RECTUS ABDOMINUS

It arises by two tendinous heads - lateral and medial from lateral part of pubic crest and anterior pubic ligament respectively. The fibres run vertically which are enclosed in the rectus sheath. It inserts into xiphoid process and 5^{th} – 7^{th} costal cartilages. The upper part is crossed by three tendinous intersections which are attached to the anterior rectus sheath and hence serve to prevent retractions of muscle in transverse incision

ANATOMY OF RECTUS SHEATH-

Rectus sheath is an aponeurotic sheath covering the rectus abdominus. It is formed by the decussating fibres from three abdominal muscles on each side, namely-external oblique, internal oblique and transversus Abdominus. It has two walls -anterior and posterior.Anterior wall is complete, and is formed by the external oblique aponeurosis, anterior lamina of aponeurosis of internal oblique upto the arcuate line. Below the arcuate line it is formed by the aponeurosis of all three muscles of abdomen.It is firmly adherent to the tendinous intersections of the rectus muscle. Posterior wall is incomplete, being deficient above the costal margin and below the arcuate line. Midway between the umbilicus and pubic symphysis, the posterior wall of rectus sheath ends in the arcuate line or lineasemilunaris or fold of douglas. Between the costal margin and the arcuate line, the posterior wall is formed by

posterior lamina of aponeurosis of internal oblique and aponeurosis of transvesus muscle. Linea alba is a tendinious raphe that extends between xiphoid process above to pubic symphysis and pubic crest below. Superficial fibres of linea alba are attached to pubic symphysis, while deep fibres are attached behind rectus abdominus to posterior surface of pubic crest. The rectus sheath serves mainly to maintain the strength of anterior abdominal wall. It also checks the bowing of rectus muscle during its contraction and thus increases the efficiency of the muscle.

FASCIA TRANSVERSALIS

It is that part of the abdomino pelvic fascia which lines the inner surface of transversusabdominis muscle. It is separated from the peritoneum by the extraperitoneal connective tissue. This fascia extends anteriorly to be continuous with its fellow of the opposite side, adherent to lineaalba.

Posteriorly it merges with the anterior layer of thoracolumbar fascia and is continuous with the renal fascia. Superiorly it is continuous with diaphragmatic fascia. Inferiorly it is attached to lateral half of inguinal ligament, inner lip of iliac crest. It forms anterior wall of femoral sheath over the femoral vessels. The integrity of the transversalis fascia is absolutely essential for the abdominal wall to prevent herniation.

FUNCTIONS OF ABDOMINAL MUSCLES

The abdominal muscles provide a firm but elastic support for the abdominal viscera against gravity. This is chiefly due to the tone of internal oblique muscle.

The oblique muscles assisted by transversus, compress the abdominal viscera and thus help in expulsive acts like micturition, defecation, vomiting and parturition. They also assist in forceful expiration, coughing, sneezing, blowing, shouting, etc.

Rectus abdominis flexes the lumbar spine, lateral flexion by oblique muscles, rotation of the trunk by combined action of the external oblique with the opposite internal oblique.

SUPERFICIAL VASCULAR SUPPLY AND DRAINAGE

Cutaneous nerves

The muscles and skin of the abdominal wall are almost entirely supplied by the lower intercostal and subcostal nerves.

Cutaneous vessels

These small arteries arise from the posterior intercostal arteries and epigastric arteries. Below the umbilicus the skin and superficial fascia are supplied by three small branches from each femoral artery.

Superficial veins

Below the umbilicus these drain with the superficial arteries to the great saphenous vein in the groin and eventually to the inferior venacava. Above the umbilicus, they pass to the axilla and so to the superior vena cava

DEEP VASCULAR SUPPLY AND DRAINAGE OF ABDOMINAL WALL

Deep arteries of the anterior abdominal wall

Anterior abdominal wall is supplied by internal thoracic and external iliac arteries. Internal thoracic artery through its terminal branches namely superior epigastric artery and musculophrenic artery. Superior epigastric artery supplies rectus muscle. Musculophrenic artery supplies diaphragm, anterior abdominal wall and 7 th-9th intercostal spaces.

External iliac artery gives off

a) inferior epigastric artery which supplies rectus muscle and ends by anastomosing with the superior epigastric artery.

b) deep circumflex iliac artery which supplies transversalis muscle.

Lymphatic drainage

The deep lymphatics above the umbilicus drain to internal mammary lymph nodes and below the umbilicus to deep iliac nodes.

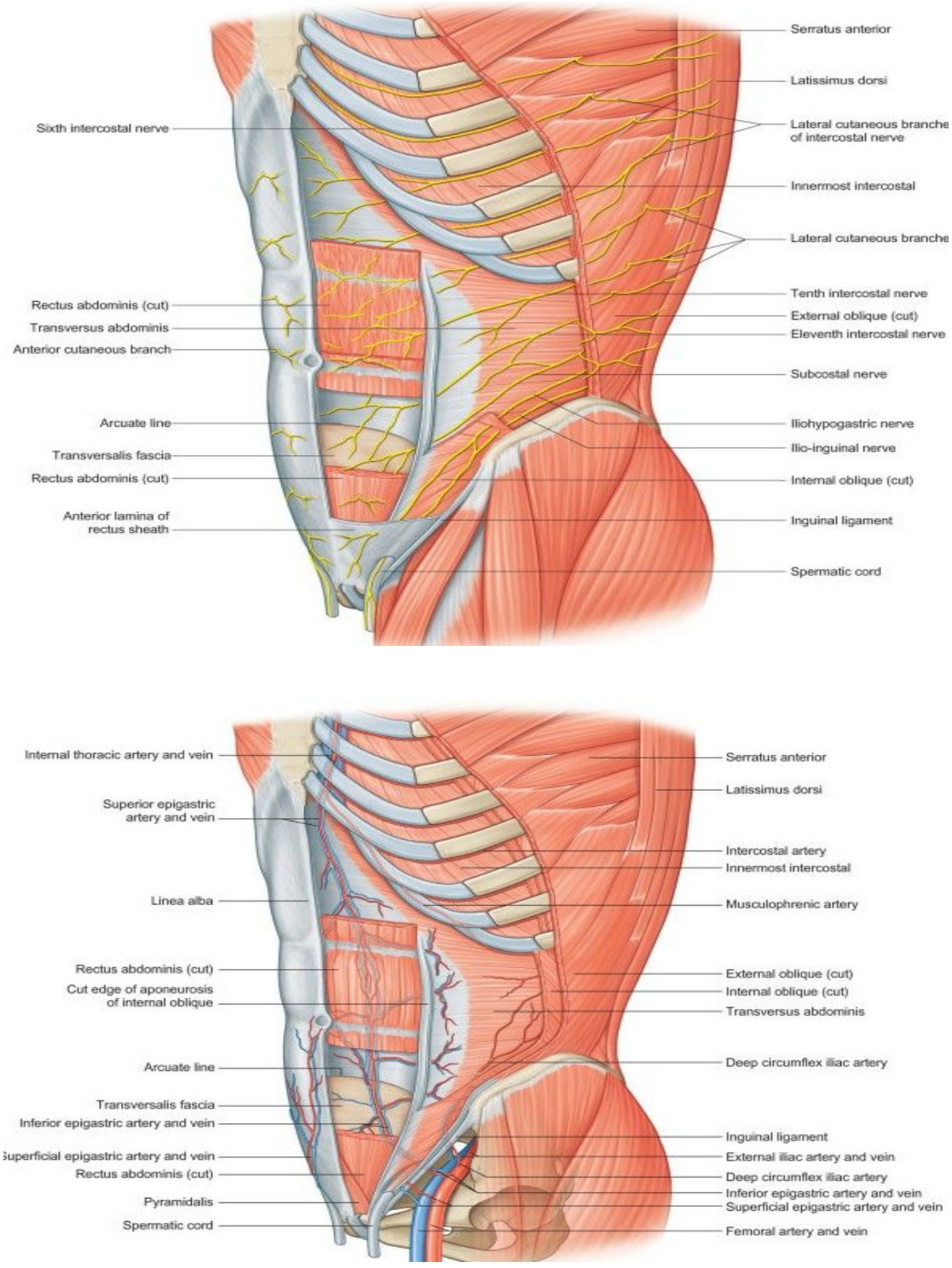


FIGURE-3: NEURO VASCULAR SUPPLY TO ANTERIOR ABDOMINAL WALL.(5)

ABDOMINAL INCISIONS(6)

An incision should be selected with the following qualifications in mind:

1. It must give ready and direct access to the source of trouble and provide adequate exposure for the operation contemplated.
2. It should be extensible in the direction that probably would be required by any increase in the magnitude of the operation.
3. It should injure the fewest possible number of motor nerves, preferably not more than one.
4. It should be capable of being securely repaired so as to leave the abdominal wall at least as strong after the operation as before.
5. It should provide an acceptable cosmetic result when possible.

It is important that an abdominal incision be made long enough to provide an adequate visualization of the operative field and uncrowded conditions for the necessary manipulations.

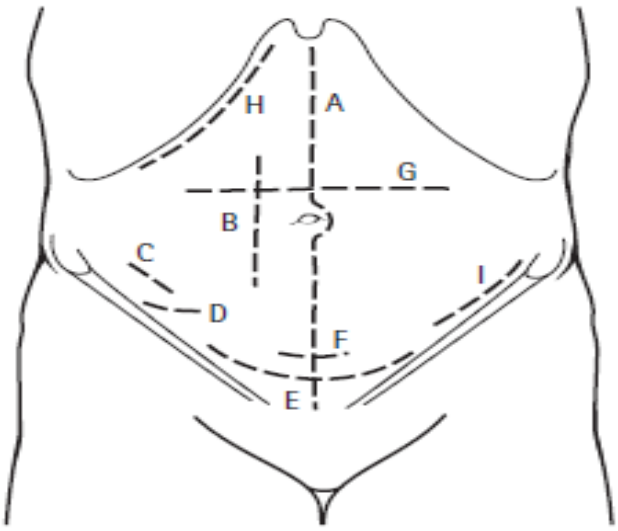


Figure 12.4 *A selection of common abdominal incisions.*
A *midline;* **B** *paramedian;* **C** *gridiron;* **D** *Lanz;* **E** *Pfannenstiel;*
F *suprapubic;* **G** *transverse upper abdominal;* **H** *subcostal 'Kocher';*
I *oblique iliac muscle-cutting.*

Different Incisions:

Midline vs. transverse incisions-(7)

- Transverse incisions in abdominal operationsare placed along the langer's lines and are based on better anatomic and surgical principles than vertical incisions.(2,3)
- Transverse incisions are more secure and less liable to cut through fascia.
- Midline incisions provide rapid access to peritoneal cavity in emergency setting with minimal blood loss.
- Midline incisions are preferred in thin patients with narrow subcostal angles to transverse incisions.

The only randomized controlled trial that has been performed that focused on the frequency of burst abdomen could find no benefit of transverse incisions(0%) over midline incisions(0.69%)(5).

However midline incision is still the most common preferred incision in general surgery. (6)

Types of abdominal incisions-

1. Dividing the fascia with no intervening muscles :

Median ,Paramedian (Rectus Retracting) ,Pararectal (Battle)

2 .Splitting Muscles :

Paramedian muscle splitting (Mayo Robson) ,Mcburney

Median incisions-

Midline supra-umbilical incision is made for the exposure of the stomach duodenum, gall bladder, Pancreas, Spleen and Hiatus. Infra-umbilical scar gives weak scar because lineaalba is narrow. This incision gives exposure to lower abdominal and pelvic viscera. Closure of the incision is in three layers.

Advantages :Linea alba is almost bloodless, no muscle fibres are divided, no nervesare injured, it affords good access to the upper

abdominal viscera and it is very quick to make and to close when speed is essential, it can be extended above and below.

Paramedian :two types- medial and lateral para median

This incision can be made on either side of midline and both supra and infra-umbilical region of the abdomen parallel to midline and one inch from it. The advantage is it gives strong scar.

The disadvantage is the longer time required for its performance and difficulty of performing the incision, when a previous laparotomy scar has to be reopened.

Lateral Paramedian Incision :

It is a modification of conventional paramedian incision in which anterior and posterior rectus sheaths are incised vertically at the junction of the middle and outer thirds of the width of rectus sheath, the muscle is reflected laterally.

Pararectal Incision of Battle :

It is employed in unilateral pelvic conditions and appendicitis. It is placed over the lateral third of the rectus in the line of its fibres below the level of umbilicus. The anterior rectus sheath is incised and rectus is displaced medially. Posterior sheath is deficient. The incision cannot be extended. Closure is same as in paramedian incision.

2.Muscle splitting incisions-

Rectus Splitting Type :(vertical muscle splitting incision)

Rectus is split longitudinally in the line of skin incision. The wound can be opened and closed quickly.

Advantage :Its value is in reopening the scar of a previous paramedian incision where dissecting the rectus muscle from the scar tissue is difficult

Disadvantage :It carries a high risk of bleeding and sectioning of nerves that may cause weakening of the corresponding area of abdominal wall

Mcburney's Grid Iron Incision :

This is commonly used for appendicectomy, the incision is made at right angles to spino umbilical line crossing at the junction of its middle and outer thirds. One third of the incision being above and two third below the line. After the skin and subcutaneous tissue, the external oblique aponeurosis is divided in the direction of its fibres, internal oblique and transversus are split and peritoneum is opened in the line of skin incision.

Lanz Incision :

The skin incision is made transversely so that it lies in the interspinous crease. There after the muscles are split as in the classical grid iron approach. It produces a less visible scar.

Oblique Muscle Cutting Incision (Rutherford Morrisson) :

The extension of the Mc Burney incision by division of oblique muscle laterally and rectus sheath medially is used on both side of iliac fossa.

Subcostal Incision (Kocher) :

Right side incision is used for the Gall bladder and Biliary tract and on left side for splenectomy.

The incision commences exactly at the middle about 2.5 to 5cm below the Xiphisternum runs outwards and downwards one inch below and parallel to costal margin. All muscles are divided in the same line. The 9^{lh} intercostal nerve should be preserved.

Transverse Incision :

These may be employed both above and below the umbilicus. In transverse incisions all the layers are divided transversely. There is no interference with the nerve supply to rectus muscles. Vertical separation of recti can be done and transversalis fascia and peritoneum incised vertically.

These transverse incisions fall in the Langer's lines hence healing is good, scar is less visible and closure is more secure.

Pfannenstiel's Incision :

It is popular for Gynaecologic operations and retropubic prostatectomy. The incision is placed in the curving interspinous crease, 5cm superior to pubic symphysis. Both anterior rectus sheaths are exposed and divided for the whole length of wound, flaps mobilized above and below.Rectus muscles are retracted laterally and the peritoneum opened vertically in the midline. An advantage is that it leaves an almost imperceptible scar. Disadvantage is that exposure afforded is somewhat limited.

Oblique Lumbar Incision (Morrrison) :

This incision is used for kidney exposure, begins in the renal angle and passes just below and parallel to the 12^{th} rib anteriorly upto the lateral border of rectus abdominis.

This incision cuts posteriorly lattissmus dorsi, serratus posterior inferior and anteriorly flat muscles of anterior abdominal wall. Peritoneum is stripped forward and the lumbodorsal fascia incised posteriorly to expose the kidney with its fascial sheath. This incision may divide lateral cutaneous branch of 12^{th} thoracic nerve and also Ilioinguinal and Iliohypogastric nerves. While closing muscles are repaired by two tiers of suture.

WOUND CLOSURE IN LAYERS(7)

Closure of the Abdominal Incision

The methods of closure are often based on local practices and the preferences of the surgeon, and the surgeon is often reluctant to change these methods later on in his or her career. Abdominal closure is performed in a multitude of fashions and there are an abundance of differently tailored studies on this matter.

The goal of wound closure is to restore function of the abdominal wall after a surgical procedure. It should leave the patient with a reasonably acceptable scar, and most importantly, it should minimize the frequency of wound dehiscence, incisional hernia, wound infection, and sinus formation.

Ideally abdominal wound are closed in 3 layers-

- Fascial closure
- Subcutaneous tissue closure
- Skin closure.

Closure of the Fascia

It has been claimed in literatures that the optimal surgical method of closing the abdominal wound is a continuous mass closure than

compared to layered closure technique. Mass closuretechnique appears to reduce the incidence of wound dehiscence, is considerably less time consuming,[18,27] is less expensive, and does not increase the incidence of incisional hernia, wound infection, or sinus formation. The choice of suture material is monofilament non absorbable suture[25,33].Numerous clinical trials have compared layered to mass abdominal closure.

Advantage-

Distribution of tension differences across the suture line and the ability of the wound to adjust to the stresses and strains of the postoperative period, which would minimize tissue strangulation and wound rupture.

Disadvantage

A single thread holds the fascia together and its breakage jeopardizes the entire wound.. Numerous clinical trials have compared layered to mass abdominal closure.

Some studies have shown an increased incidence of burst abdomen and incisional hernia with layered closure,[13-15] and some studies show no difference in these complications,[16] but no studies demonstrate an advantage of layered over mass closure.

Subcutaneous Tissue Closure

A series of simple, interrupted, absorbable polyglactic acid (vicryl) sutures to reapproximate the subcutaneous layer. These stitches are inverted to bury the knots within the wound.this may reduce the chance of dead space and further Seroma collection predisposing to wound infection.

A separate trial confirmed the reduction in wound disruption in wounds with greater than 2 cm of tissue.[31]

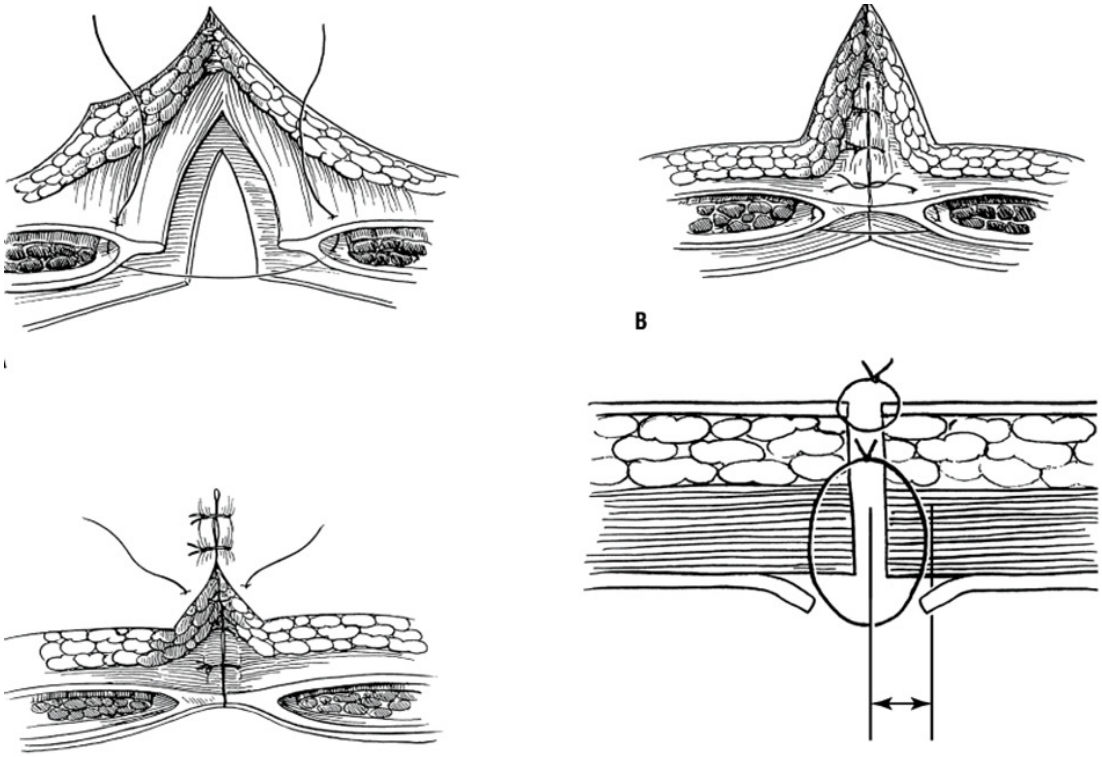


FIGURE 4: STAGES IN MASS CLOSURE OF MIDLINE ABDOMINAL INCISION

Skin Closure

A number of closure techniques for clean (class I) and clean-contaminated (class II) wounds are available for the skin.If the surgical site is heavily contaminated (class III or class IV wound), the skin should be left open to heal by secondary intention or by delayed primary skin closure.[34]. Goals of skin closure are tissue approximation, minimizing wound infection, acceptable cosmesis, and minimizing postoperative pain.

It is concluded that closure of the peritoneum is unnecessary and not recommended. It is associated with a slightly longer operative time and more postoperative pain, and there are some suggestions that it may cause increased formation of adhesions.[12]

STAGES OF WOUND HEALING[8]-

Wound healing is a complex biological process that consists of the following phases-

Broadly divide into four phases-

1. Hemostasis.
2. Inflammatory phase.
3. Proliferative phase.
4. Maturation phase.

Phase	Cellular and Bio-physiologic Events
Hemostasis	1. vascular constriction 2. platelet aggregation, degranulation, and fibrin formation (thrombus)
Inflammation	1. neutrophil infiltration 2. monocyte infiltration and differentiation tomacrophage 3. lymphocyte infiltration
Proliferation	1. re-epithelialization 2. angiogenesis 3. collagen synthesis 4. ECM formation
Remodeling	1. collagen remodeling 2. vascular maturation and regression

1. **Hemostasis-**

It begins immediately after wounding and includes vascular constriction and fibrin clot formation. The clot and the surrounding wound tissue release pro-inflammatory cytokines and growth factors such as transforming growth factor(TGF)-β,platelet derived growth factor(PDGF), fibroblast growth factor(FGF), and epidermal growth factor(EGF). These factors promote infiltration of inflammatory cells described in the next phase.(9)

2. **Inflammatory phase**-

This is characterized by sequential infiltration of neutrophils, macrophages and lymphocytes.

Neutrophils help in clearance of invading microbes and cellular debris in the wound area, although these cells also produce substances such as proteases and reactive oxygen species, which cause additional bystander damage. Macrophages in the early wound, release cytokines that promote the inflammatory response by recruiting and activating additional leucocytes. They are also responsible for inducing and clearing apoptotic cells(including neutrophils), thus resulting in resolution of inflammation. They undergo a phenotypic transition to a reparative state that stimulates keratinocytes, fibroblasts and angiogenesis to promote tissue regeneration(10).in this way, macrophages promote the transition to the proliferative phase of healing.

Lymphocytes peak during the late proliferative/early remodeling phase. Role of T lymphocytes is still not clearly understood.Several studies suggest that delayed T-cell infiltration along with decreased T-cell concentration in the wound site is associated with impaired wound healing, while others have reported that CD 4+cells (T-helper cells) have a positive role in wound healing and CD8+ cells (T-suppressor-cytotoxic cells) play an inhibitory role in wound healing (Swift *et al.*, 2001; Park and Barbul, 2004).

3. **Proliferative phase**-

It generally overlaps with the inflammatory phase and is characterized by epithelial proliferation and migration over the provisional matrix within the wound(re-epithelialization). Fibroblasts and endothelial cells are the predominant cells types present and support capillary growth, collagen formation, and the formation of granulation tissue at the site of injury. within the wound , fibroblasts produce collagen as well as glycosaminoglycans and proteoglycans, which are major componenets of extracellular matrix.

4. **Remodeling**

The wound healing enters the final remodeling phase. This phase can last for years.in this phase, there happens regression of many of the newly formed capillaries. One important feature of the remodeling phase is ECM remodeling to anarchitecture that approaches that of the

normal tissue. The woundalso undergoes physical contraction throughout the entire wound healingprocess, which is believed to be mediated by contractilefibroblasts (myofibroblasts) that appear in the wound (Gosain and

DiPietro, 2004; Campos *et al*., 2008).

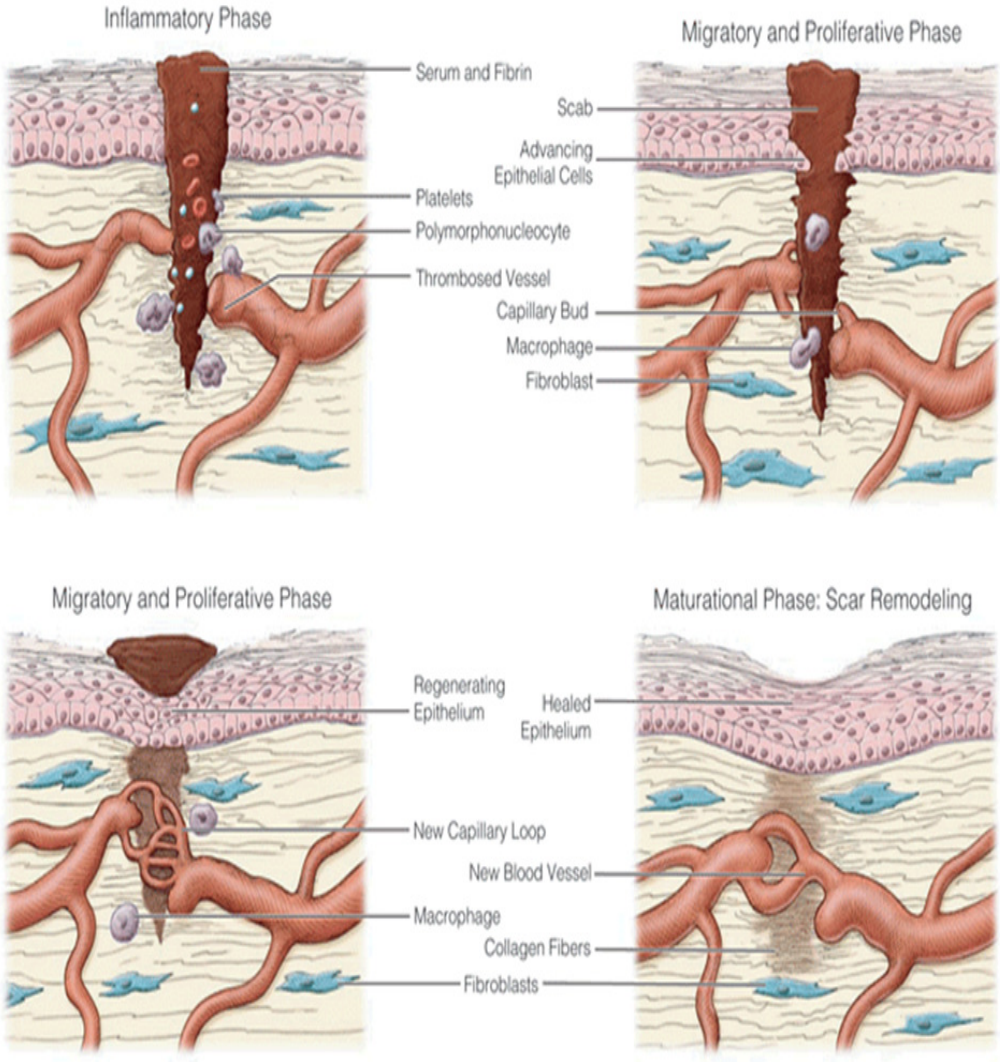


FIGURE 5: STAGES OF WOUND HEALING

FACTORS AFFECTING WOUND HEALING-

Local Factors	Systemic Factors
. Oxygenation . Infection . Foreign body . Local tension.	. Age and gender . Sex hormones. . Diseases: diabetes, hereditary healing disorders, jaundice, uremia. . Obesity. . Medications: glucocorticoid steroids, non-steroidal anti-inflammatory drugs, chemotherapy . Alcoholism and smoking . Psychological stress. . Immunocompromised conditions: cancer, radiation therapy, AIDS. . Malnutrition.

LOCAL FACTORS-

- **Oxygenation**-Hypoxia signals macrophages, keratinocytes, and fibroblasts to produce that include PDGF, TGF-β, VEGF, tumor necrosis factor-α (TNF-α), and endothelin-1, that promote cell proliferation,It prevents wounds from infection, inducesangiogenesis, increases keratinocyte differentiation, migration,and re-epithelialization, enhances fibroblast proliferation andcollagen synthesis, and promotes wound contraction,migration and chemotaxis, and angiogenesis in wound healing.(11)
- **Infections** - Invasive infection is defined as the presence of replicating organisms within a wound with subsequent host injury.Both bacteria and endotoxins can lead to the prolonged elevation of pro-inflammatory cytokines such as interleukin-1 (IL-1) and TNF-α and elongate the inflammatory phase. If this continues, the wound may enter a chronic state and fail to heal. This prolonged inflammation also leads to an increased level of matrix metalloproteases (MMPs), a family of proteases that can degrade the ECM.Mature biofilms develop protected microenvironments and are more resistant to conventional antibiotictreatment. *Staphylococcus aureus*(*S. aureus*), *Pseudomonas*

aeruginosa(*P. aeruginosa*), and β -hemolytic *streptococci* are common bacteria in infected and clinically non-infected wounds.(12)

- **Foreign body**- clot, necrotic debris,dirt,glass prolong the inflammatory phase, leads to increased susceptibility for infection and prolonged re-epithelialisation.
- **Local tension** -Wound tension increases tissue pressure, reducing microperfusion and the availability of oxygen to the wound resulting in wound dehiscence.

SYSTEMIC FACTORS –

- **Age**- increasing age is a major risk factor for impaired wound healing.Delayed wound healing in the aged is associated with an altered inflammatory response, such as delayed T-cell infiltration into the wound area with alterations in chemokine production and reduced macrophage phagocytic capacity.
- **Gender** -Estrogen affects wound healing by regulating a variety of genes associated with regeneration, matrix production, protease inhibition, epidermal function, and the genes primarily associated with inflammation.While androgens regulate cutaneous wound healing negatively.(13)
- **Systemic diseases**–

a. Diabetes- Hyperglycemia leads to

i. Adds to the oxidative stress when the production of ROS exceeds the anti-oxidant capacity, as a result of prolonged inflammatory response(14).

ii. Formation of advanced glycosylation end products(AGEs) their interaction with the receptors(RAGE)are associated with impaired wound healing.

iii. High levels of metalloproteases are a feature of diabetes,This increased protease activity supports tissue destruction and inhibits normal repair processes.

b. Hereditary healing disorders-hereditary disorders of connective tissues (HDCTs) encompass a spectrum of conditions linked patho-physiologically by abnormalities of collagen, fibrillin, and matrix proteins manifest with poor wound healing.

c. Uremia- Uremia can interfere with wound healing by slowing granulation tissue formation and inducing the synthesis of poor quality collagen.

d. Jaundice- it has been shown that prolyl hydroxylase activity, which reflects the rate of collagen synthesis, is decreased in the skin of patients with obstructive jaundice

- **Obesity** - Obese individuals frequently face wound complications, including skin wound infection, dehiscence, hematoma and seroma formation.

- **Medications** –

a. Glucocorticoid steroids- inhibit wound repair *via* global anti-inflammatory effects and suppression of cellular wound responses, including fibroblast proliferation and collagen synthesis.Also inhibit production of hypoxia-inducible factor-1 (HIF-1), a key transcriptional factor in healing wounds.(15)

b. Non-steroidal anti-inflammatory drugs(NSAIDS)- There are few data to suggest that short-term NSAIDs have a negative impact on healing. However, the question of whether long-term NSAIDs interfere with wound healing remains open.

c. Chemotherapeutic drugs- inhibit cellular metabolism, rapid cell division, and angiogenesis and thus inhibit many of the pathways that are critical to appropriate wound repair.In addition, these agents weaken the immune functions and increase the risk of wound infection.

- **Alcohol consumption**- Studieshave demonstrated profound effects of alcohol on host-defensemechanisms, although the precise effects are dependent uponthe pattern of alcohol exposure (*i.e*., chronic *vs*. acute alcoholexposure, amount consumed, duration of consumption, timefrom alcohol exposure, and alcohol withdrawal).The most significant impairment seems to be in wound angiogenesis, which is reduced by up to 61% following a single ethanol exposure.(16)

- **Smoking** –

i. Nicotine probably interferes with oxygen supply by inducing tissue ischemia, since nicotine can cause decreased tissue blood flow *via* vasoconstrictive effects.(17)

ii. Causes impaired white blood cell migration, resulting in lower numbers of monocytes and macrophages in the wound site, and reduces neutrophil bactericidal activity.

- **Malnutrition** –

i. Glucose is the major source of fuel used to create the cellular ATP that provides energy for angiogenesis and deposition of the new tissues.

ii. Deficiency of protein can impair capillary formation, fibroblast proliferation, proteoglycan synthesis, collagen synthesis, and wound remodeling.

iii. Vitamins C (L-ascorbic acid), A (retinol), and E (tocopherol) show potent anti-oxidant and anti-inflammatory effects. Vitamin C has many roles in wound healing linked to collagen synthesis , fibroblast proliferation and capillary fragility.. Also, vitamin C deficiency leads to an impaired immune response and increased susceptibility to wound infection.(18) In summary, proteins, carbohydrates, arginine, glutamine, polyunsaturated fatty acids, vitamin A, vitamin C, vitamin E, magnesium, copper, zinc, and iron play a significant role in wound healing, and their deficiencies affectwound healing.

TYPES OF WOUND HEALING-(19)

1. PRIMARY INTENTION
2. SECONDARY INTENTION.
3. TERTIARY INTENTION.

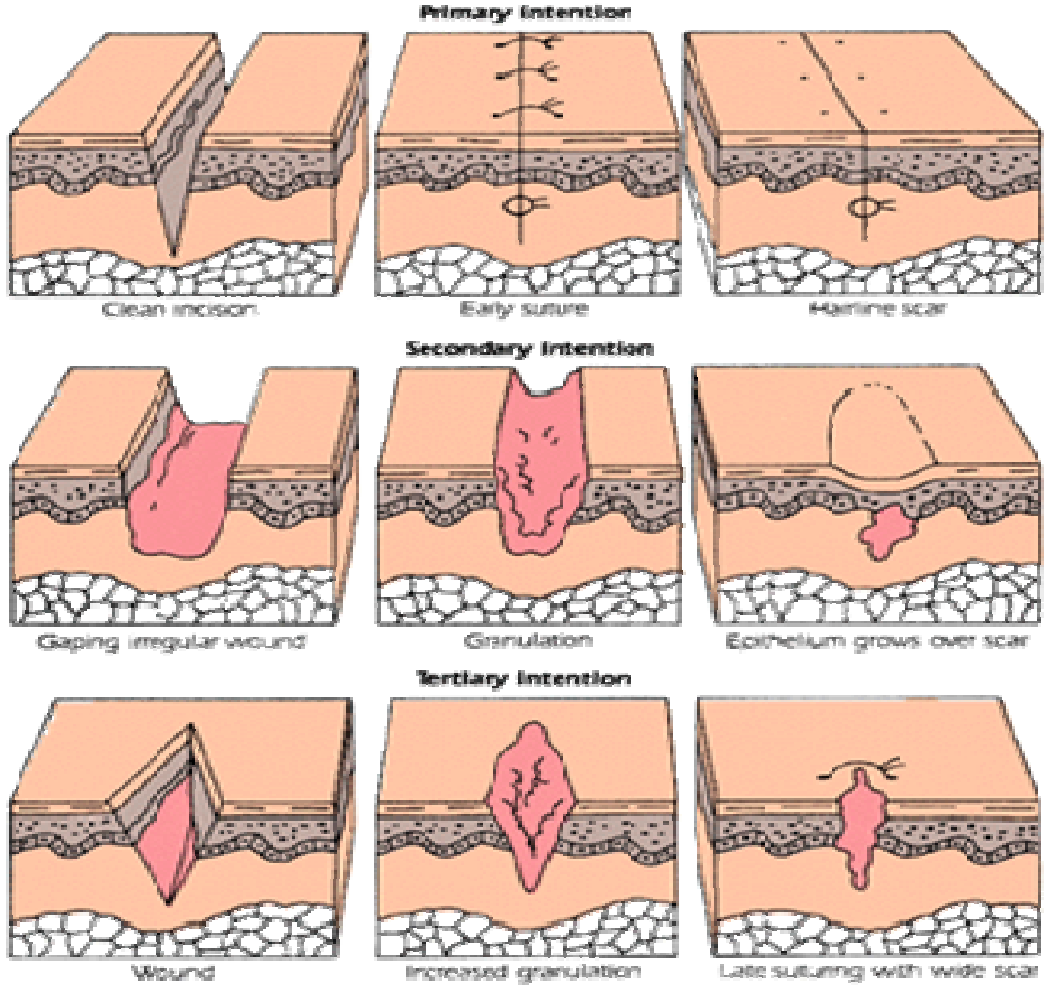


FIGURE 6- TYPES OF WOUND HEALING

HEALING BY PRIMARY INTENTION- (primary union)

a) Clean incised surgical wounds

b) Tension free approximation of skin edges.

c) Wound is treated within 24 h following injury, prior to development of granulation tissue.

d) they are often the fastest and most cosmetically pleasing method of healing.

HEALING BY SECONDARY INTENTION-(granulation)

a) a wound left open and allowed to close by epithelialization and contraction.

b) Commonly used in the management of contaminated or infected wounds.

c) Wound is left open to heal without surgical intervention.

d) Complications include late wound contracture and hypertrophic scarring.

HEALING BY TERTIARY INTENTION-(delayed primary closure)

a) Indicated for infected or unhealthy wounds with high bacterial content, wounds with a long time lapse since injury, or wounds with a severe crush component with significant tissue devitalization.

b) Often used for infected wounds where bacterial count contraindicates primary closure and the inflammatory process can be left to debride the wound.

c) The wound is left open for 4-5 days for observation, over this time the inflammatory process has reduced the bacterial concentration of the wound to allow safe closure.

d) Wound edges are approximated within 3-4 days and tensile strength develops as with primary closure.

CLASSIFICATION OF SURGICAL WOUNDS-(19)

CATEGORY	CRITERIA	INFECTION RATE
Clean	No hollow viscus entered; Primary wound closure; No inflammation; Nobreaks in aseptic technique,elective procedure.	1%-3%
Clean-contaminated	Hollow viscus entered but controlled no inflammation; primary wound closure, minor break in aseptic technique. Mechanical drain used, bowel preparation pre-operatively.	5%-8%
Contaminated	Uncontrolled spillage from viscus; non purulent inflammation apparent; open traumatic wound; major break in aseptic technique.	20-25%
Dirty	Untreated, uncontrolled spillage from viscus;pus in operative wound; open suppurative wound severe inflammation.	30%-40%

ABDOMINAL WOUND DEHISCENCE (BURST ABDOMEN)

It refers to the post-operative separation of the abdominal musculo-aponeurotic layers. It is complete, when all the layers of the abdominal wall have burst apart with or without associated protrusion of a viscus(evisceration). It is among the most dreaded complication faced by surgeons.

ETIO-PATHOGENESIS AND RISK FACTORS-

In addition to certain specific factors all those conditions which affect wound healing in general are included in the Etio-pathogenesis. Broadly classified into- local and systemic factors

LOCAL FACTORS	SYSTEMIC FACTORS
Incisions Techniques of laparotomy closure. Wound infection, hematoma and Seroma. Intra-abdominal infection. Elevated intra-abdominal pressure. Pervious wound dehiscence. Emergency surgery.	Advanced age Systemic diseases (diabetes, uremia, ascites, anemia, chronic obstructive pulmonary diseases (COPD). Obesity. Drugs- steroids, chemotherapy. Malnutrition.

LOCAL FACTORS-(7)

- **INCISCIONS-**

a) Transverse incisions are superior to vertical incision with regard to long term and short term outcomes including burst abdomen and incisional

hernia. However, the vertical incision is still the most commonly performed incision in general surgery. The only randomized controlled trial that has been performed focused on frequency of burst abdomen could find no benefit of transverse incision(0%) over midline incision(0.69%).

b) The advantages of Para median incision have been investigated in the prospective randomized trials demonstrating that the conventional Para median incision offers no advantage in wound failure rates when compared to midline or transverse incision.

- **TECHNIQUE OF LAPROTOMY CLOSURE-**

a) Several randomized trials have shown that technique of mass closure with a running suture is the best method for closure of midline wounds.

b) Sutures may rupture because it is too weak for the tension to be placed upon.it may cut through the tissues either because sutures are placed too close to the wound edge or because of excessive weakening of tissues.

c) Another frequent technical error is improper knot tying which may lead unraveling.

A general guideline is to place suture 1 cm apart with 1 cm bites of fascia.

WOUND INFECTION-

a) *Wound infection* is the discharge of pus from a primarily closed wound, or evidence of soft tissue necrosis or cellulitis about a wound that remains open for delayed or secondary closure.

b) *Necrotizing fasciitis* occurs when the rectus or lateral abdominal wall investing fascia becomes necrotic and undergoes dissolution from invasive infection arising from the surgical wound of the abdominal wall.

c) SURGICAL SITE INFECTIONS(SSI)-(7)

Incisional SSIs are further divided into those involving only skin and subcutaneous tissue (superficial incisional SSI) and those involving deeper soft tissues of the incision (deep incisional SSI). Organ/space SSIs involve any part of the anatomy (e.g., organ or space) other than incised body wall layers, that was opened or manipulated during an operation.

Superficial Incisional Surgical Site Infection

Infection within 30 days after the operation and only involves skin and subcutaneous tissue of the incision and at least one of the following:

1. Purulent drainage with or without laboratory confirmation, from the superficial incision.

2. Organisms isolated from an aseptically obtained culture of fluid or tissue from the superficial incision.

3. At least one of the following signs or symptoms of infection: pain or tenderness, localized swelling, redness, or heat and superficial incision is deliberately opened by surgeon, unless incision is culture-negative.

Deep Incisional Surgical Site Infection

Infection occurs within 30 days after the operation if no implant is left in place or within one year if implant is in place and the infection appears to be related to the operation and infection involves deep soft tissue (e.g. fascia, muscle) of the incision and at least one of the following:

1. Purulent drainage from the deep incision but not from the organ/space component of the surgical site.

2. A deep incision spontaneously dehisces or is deliberately opened by a surgeon when the patient has at least one of the following signs or symptoms: fever (>38°C), localized pain or tenderness, unless incision is culture-negative.

3. An abscess or other evidence of infection involving the deep incision is found on direct examination, during reoperation, or by histopathology or radiologic examination

Organ/Space Surgical Site Infection

Infection occurs within 30 days after the operation if no implant is left in place or within one year if implant is in place and the infection appears to be related to the operation and infection involves any part of the anatomy (e.g., organs and spaces) other than the incision which was opened or manipulated during an operation and at least one of the following:

1. Purulent drainage from a drain that is placed through a stab wound into the organ/space.
2. Organisms isolated from an aseptically obtained culture of fluid or tissue in the organ/space
3. An abscess or other evidence of infection involving the organ/space that is found on direct examination, during reoperation, or by histopathology or radiologic examination.

DIAGNOSIS-(7)

Three patterns of occurrence of abdominal wound dehiscence are noted-

- Abrupt rush of pink serosanguineous discharge from the wound with a soft and boggy swelling noted underneath the skin following which wound exploration is done.

- Sudden give away in the wound following excessive strain resulting in dehiscence.
- Delayed onset of dehiscence in a setting of wound infection.

The seepage of serosanguineous fluid through a closed abdominal wound usually around 8^{th} to 10^{th} post- operative day is an early sign of abdominal wound dehiscence with possible evisceration. When this occurs, the surgeon should remove one or two sutures in the skin and explore the wound manually, using a sterile glove. If there is separation of the rectus fascia, the patient should be taken to the operating room for primary closure. Wound dehiscence may or may not be associated with intestinal evisceration. When the latter complication is present, the mortality rate is dramatically increased and may reach 30%.

TREATMENT[7]**-**

The basic principles of management of abdominal wall dehiscence and evisceration are early diagnosis and surgical closure.The basic treatment principle for repair of the disrupted wound is re-suturing of wound edges. The treatment modality depends on the size of the defect and general condition of the patient.

The objective of surgery is to replace the eviscerated organs into the abdominal cavity, and to prevent recurrent dehiscence and later

development of ventral hernias. As soon as the condition is recognized, the wound and protruding viscera should be freely bathed with warm normal saline solution and covered with large sterile dressing. Patient should be moved to the operating room and general anesthesia should be administered and a nasogastric tube placed to decompress the stomach. The edges of the abdominal wall are then lifted upward, and the prolapsed bowel is replaced back into peritoneal cavity. At this stage, fragments of suture material are removed, and the wound edges are freshened by debriding away necrotic tissue and edematous skin tags. It will be noted in many cases that the fascial edges are swollen and retracted outward, highlighting the need for immediate repair of the dehisced wound to prevent further retraction of the wound edges. If only a very small area of the wound has been disrupted, this portion alone should be sutured. However, if more than half of the wound has been disrupted, the correct procedure is to open the remaining portion of the wound and suture the whole wound afresh

OPERATIVE PROCEDURE-

- Resuturing should be performed using a strong monofilament non-absorbable suture such as polypropylene.
- Retention sutures, if placed, can be internal (wide bites of the fascia deep to the skin), or external (full-thickness bites of fascia and skin are taken together).
- External retention sutures consists of placing strong monofilament nylon stitches 2.5 cm from the margin of the wound and about 2.5 cm apart that transfix all the layers of the abdominal wall on both margins of the wound. As they are introduced, the free ends are clipped with hemostats. The fascia is then closed with a running non-absorbable suture before the retention sutures are threaded though 5-cm pieces of protective rubber tubing and firmly tied.
- If the size of defect is small, the patient is critically ill, and there is no evisceration of viscera, then the dehiscence can be dealt with conservatively to heal by secondary intention. This will result in the development of an incisional hernia that can be repaired later, after the patient is discharged. This technique involves packing the open wound with a moist sterile dressing and generous strips of elastic adhesive tape placed transversely across the abdomen

POST-OPERATIVE CARE

- Patient should avoid excessive physical activity.
- All the comorbid factors like wound infection ,cough, diabetes, anemia should be identified and appropriately treated.
- In case of an open wound planned for managing conservatively, wound should be packed with moist saline dressings and changed at regular intervals until healing takes place by formation of granulation tissue.
- Patient should be given high protein diet necessary for wound healing.
- Retention sutures are generally kept in place for 2-4 weeks.
- Abdominal binder may be used for additional support.
- Recent introduction of vacuum-assisted wound closure devices (VAC devices) has been helpful in such cases. By applying such dressings the wounds are kept clean, and the controlled negative pressure helps to evacuate fluid and stimulate healing.

COMPLICATIONS

Occurrence of wound dehiscence should be considered a red flag sign indicating following conditions. The conditions might occur following dehiscence or may be the underlying factor in causation.

- Wound sepsis.
- Intra-abdominal abscess.
- Bowel fistulae

- Late complications that develops in the scar of the surgical incision - incisional hernia.

INNOVATIVE TECHNIQUES IN MANAGEMENT OF WOUND DEHISCENCE

1. **VACCUM ASSISTED WOUND CLOSURE DEVICES**(7)-uses a closed suction system to remove the exudate and the infected material from the open abdominal wound and stimulate healing

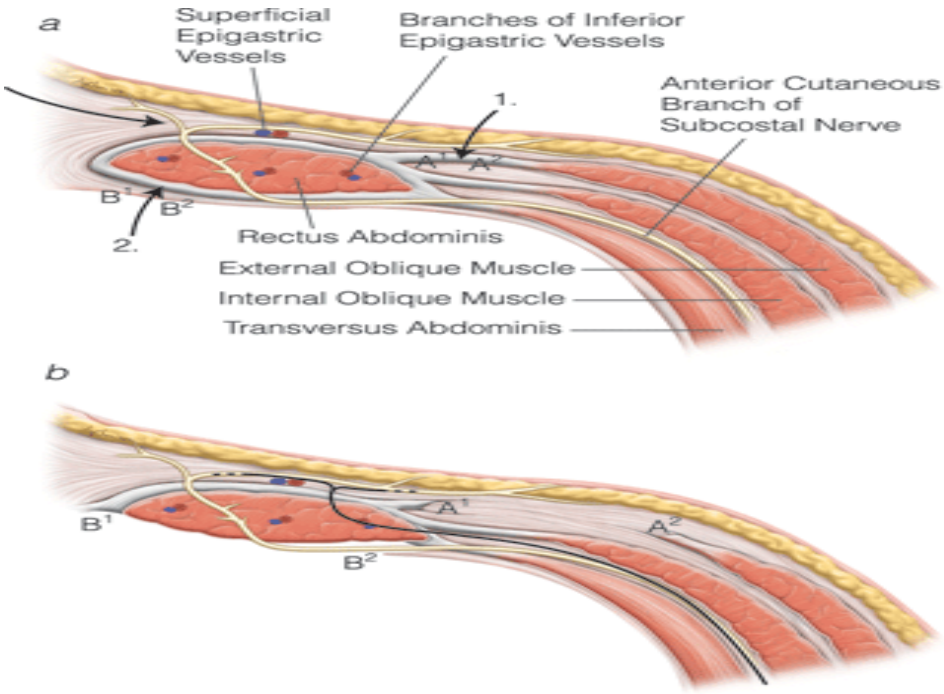
- . The entire wound and surrounding skin are covered with an adhesive transparent membrane, which is perforated by a drainage tube attached to the suction system.
- This applies negative pressure and prevents escape of fluid, because the membrane adheres to the skin all the way around the wound.
- Fluid within the wound is absorbed by the foam sponge and removed via the drainage tube into a container attached to the suction unit.
- Duration of therapy is between 2 and 21 weeks with a mean of 5 weeks.

- Complications include partial skin loss and entero-cutaneous fistula formation in few cases.

2. **COMPONENT SEPERATION TECHNIQUE-**[20,21]It is a type of rectus abdominis muscle advancement flap, was first used to reconstitute the linea alba, reduce abdominal wall tension, and provide a dynamic abdominal wall in patients with large abdominal wall defects .

Operative technique-

- The skin is opened via a midline incision. If a skin defect exists or if the intestine is covered with a split-thickness skin graft, the abdominal cavity is entered via an incision just lateral to the defect. The intestine andother viscera are dissected free from the ventral abdominal wall.
- The skin and subcutaneous fat are dissected free from the anterior rectus sheath and the aponeurosis of the external oblique muscle to about 5 cm lateral to the lateral border of the rectus sheath.
- The aponeurosis of the external oblique muscle is incised 1 to 2 cm lateral to the lateral border of the rectus abdominis muscle.
- The myoaponeurosis of the external oblique muscle is transected longitudinally over its full length extending over the thoracic wall upto 10 cm.



- The rectus abdominis muscle can be shifted medially maximally in the upper abdomen by dissecting free from its attachment to ribs.
- The external oblique muscle is separated from the internal oblique muscle in the avascular plane between both muscles to the midaxillary line.
- A further gain of 2 to 4 cm can be achieved by separating the posterior sheath of the rectus abdominis muscle.
- The abdominal wall is closed in the midline with a running suture of a non-absorbable or slowly absorbable suture material, taking big bites of fascia.Suction drains are placed subcutaneously, and the subcutaneous tissue and skin are closed.
- Defects up to 28 cm in the waistline can be bridged in this way.

ADVANTAGES-

- Allows reconstruction of a large defect of about 25-30cms without requiring a free distant transposition flap.
- Restores structural support of the abdominal wall, provides stable soft tissue coverage, and optimizes esthetic appearance of complex abdominal wall defects.
- As it does not include the use of mesh it is used in repair of contaminated wounds as well.

DISADVANTAGES-

- Sensory disturbance over the abdominal wall.
- Hematoma and Seroma formation.
- Skin necrosis.
- Recurrence rate of hernia was reported to be 30%.

3. **DYNAMIC PARIETAL CLOSURE**(22)-

It is a simple, quick, inexpensive, and compatible procedure in which silicone loop sutures are used to strengthen a conventional aponeurotic closure.It has the advantages but not the disadvantages of the use of retention sutures.

- successive mass closures were performed along the entire length of the median laparotomy with elastic silicone loops.
- Each loop was placed trans-fascially across the wound to obtain a U-

shaped suture every 4 cm.

- Finally the dynamic parietal closure's tension was adjusted, and compresses were placed between the skin and the loops
- The system was removed after 21 days.

ROLE OF PROPHYLACTIC RETENTION SUTURES-(7)

The purpose of using retention sutures is to relieve tension along the primary suture line to prevent wound disruption and allow normal relaxed wound healing.

They are sometimes employed for initial laparotomy closure when poor wound healing is anticipated, as in obese, cirrhotic, and cachectic patients, those receiving corticosteroids, or when increased intra-abdominal pressure is anticipated, as in postoperative ileus.

Only one randomized trial of full-thickness retention suture placement in midline laparotomy closure over standard mass closure has been done ,which could not identify any benefit of retention sutures.

DISADVANTAGES-

- Include the potential hazard of caught viscera, significant postoperative pain, a residual cross-hatched scar, and leakage of intraperitoneal fluid through the wound.
- For these reasons, retention suture closure has largely fallen out of favor by many surgeons during initial laparotomy closure.

REVIEW OF SCORING SYSTEM TO PREDICT ABDOMINAL WOUND DEHISCENCE

Several studies have been performed to identify the risk factors for development of wound dehiscence. But very few have developed a scoring system based on the risk factors in predicting the probability of wound dehiscence.

ABDOMINAL WOUND DEHISCENCE RISK INDEX:[1]

The largest study which analyzed over 570 cases of dehiscence in the veterans affair population. Peri-operative data from 17,044 laparotomies resulting in 587(3.4%) wound dehiscence performed at 132 veterans affairs medical center between October 1 1996 and September 30 1998 were used to develop the model. Data from 17,763 laparotomies performed between October 1998 and September 30,2000 ,resulting in 562(3.2%) dehiscence's were used to validate the model. Data were subjected to multivariate analysis and resulted in identification of several independent risk factors. The model was used to create a scoring system, designated the abdominal wound dehiscence risk index for prediction of occurrence of post operative dehiscence.

RISK FACTOR	SCORE
1. Cerebro vascular accident without deficit.	**4**
2. History of chronic obstructive Pulmonary disease.	**4**
3. Current pneumonia	**4**
4. Emergency procedure	**6**
5. Operative time >2.5hrs	**2**
6. Postgraduate year 4 as a surgeon	**3**
7. Clean wound classification	**-3**
8. Superficial wound infection	**5**
9. Deep wound infection	**17**
10. Failure to wean	**6**
11. One or more complications	**7**
12. Return to operating room on stay	**-11**

Risk categories for wound dehiscence

RISK CATEGORY	TOTAL SCORE	PREDICTED RATE OF DEHISCENCE
Low	**<3**	**1.47%**
Medium	**4-10**	**2.70%**
High	**11-14**	**4.53%**
Very high	**>14**	**10.90%**

-

The main drawback of this study includes the data collected from veterans carry more comorbidities than the general population, therefore results cannot be applied to the general population and criticized for the lack of validation.

ABDOMINAL WOUND DEHISCENCE RISK MODEL(23)

In this case control study by van ramshorst et al conducted at the dept. of general surgery ,Erasmus university medical center, Rotterdam, Netherlands a risk model was developed to recognize the high risk patients and to identify the independent risk factors for abdominal wound dehiscence.

Data collected from medical registries from January 1985 to December 2005 of all open abdominal surgeries regarding the patient

peri operative factors. Excluded surgeries were laparoscopic, gynecological, urological surgeries, appendectomies, and ventral hernia. Each case was matched with three controls selected by systemic random sampling. A total of 363 cases and 1089 controls were analyzed. Factors not matched were age, sex,type of surgery which were reported as the risk factors and had to be analyzed. Controls were selected from group of patients who had undergone open abdominal surgery from Sunday midnight to Friday midnight to avoid excessive inclusion of emergency operations.

Patients were compared with controls using chi-sqaure test or mann-whitney U test for categorical or continuous data.

Subsequently, multivariate stepwise logistic regression with backwards elimination was used to identify major independent factors of abdominal wound dehiscence.

The resulting regression coefficients for the major risk factors were used as weights for these variables to calculate a risk score for abdominal wound dehiscence.

RISK SCORE FOR ABDOMINAL WOUND DEHISCENCE

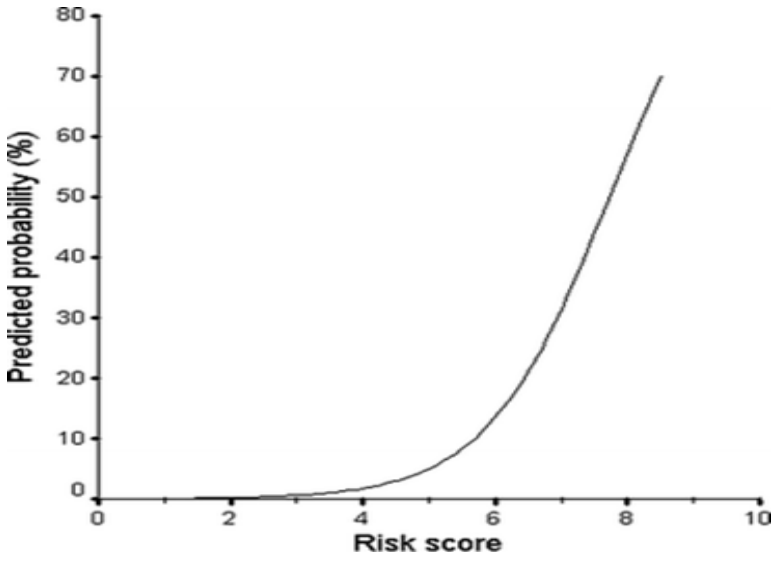
VARIABLE	RISK SCORE
1. Age category, years	
40-49 yrs	**0.4**
50-59 yrs	**0.9**
60-69yrs	**0.9**
>70 yrs	**1.1**
2. Male gender	**0.7**
3. Chronic pulmonary disease	**0.7**
4. Ascites	**1.5**
5. Jaundice	**0.5**
6. Anemia	**0.7**
7. Emergency surgery	**0.6**
8. Type of surgery	
Gall bladder/bile duct	**0.7**
Esophagus	**1.5**
Gastroduodenum	**1.4**
Small bowel	**0.9**
Large bowel	**1.4**
Vascular	**1.3**
9. Coughing	**1.4**
10. Wound infection	**1.9**

Logistic regression analysis of abdominal wound dehiscence in relation to calculated risk scores showed that increase in risk score by one point is associated with an increase of risk of abdominal wound dehiscence by 2.96($P<0.001$).

The fit of the model was good, as shown by the hosmer and lemeshowtest($P=0.79$).

The predictive value of risk model was assessed by plotting the sensitivity versus the fraction false positives for all possible cut off values in a receiving operating characteristic curve(ROC curve) which showed 0.91 ,showing a high predictive value of the risk score.

The absolute risk of developing wound dehiscence in relation to the risk score is shown below-



The mean probability per risk score category is shown below-

Table 5 Absolute risk of abdominal wound dehiscence in the validation population by risk score

Risk score	Total no. of patients	Percent	Abdominal wound dehiscence		Mean probability[b] (%)
			Number	Percent	
0–2	188	27.4	0	0.0	0.1
2–4	329	48.0	2	0.6	0.7
4–6	138	20.1	8	5.8	5.5
6–8	29	4.2	7	24.1	26.2
>8	2	0.3	2	100	66.5
Total	686	100	19	2.8	

[a] Observed percentages within risk score groups

[b] Mean values of individual calculated probabilities according to risk score within risk score group

The calculation of probability of wound dehiscence for an individual surgical patient is performed in two steps-

First , the total risk score is calculated by adding the weights of various variables

In the next step ,the probability of wound dehiscence, P, is calculated according to logistic formula:

$$P= ex/ (1+ex)*100\%;$$

Where ‘ex’ represents the exponential function and ‘x’ represents ‘-8.37 + (1.085 x calculated total risk score)’.

PECULIARITIES IN THE STUDY-

1. Diseases of Liver, kidney and pancreas did not show any proven risk factors and the effects of these variables did not significantly differ from each other, therefore were not included in the risk category.

2. Risk factors that did not have independent effects included hypertension, uremia, corticosteroid abuse, diabetes ,previous laparotomy, malignancy, sepsis and post-operative vomiting , although have been identified as risk factors by several authors.

3. Jaundice was found to be an independent risk factor, though it has not been confirmed by other studies.

4. Studies are needed to evaluate other possible factors such as nutritional state where limited retrospective data are available.

USES OF PROGNOSTIC SCORING SYSTEM-

1. Identification of high risk patients and to plan intervention strategies which involve preventive wound closing with such reinforcements as mesh (biologic).
2. Highlights the role of a surgeon in minimizing the wound dehiscence by practice of optimal surgical techniques in preventing wound dehiscence.
3. The peri-operative factors contributing to dehiscence can be mitigated in reducing the occurrence of dehiscence.
4. Results of such studies will lead to better, evidence based treatment options for abdominal wound dehiscence and eventually, a lower incidence of this severe complication.

MATERIALS AND METHODS

DESIGN-

Prospective observational study.

METHODOLOGY-

100 patients admitted in general surgery department in Coimbatore Medical College undergoing abdominal laparotomy, will be studied prospectively between September 2012 to November 2013.

Inclusion criteria:

- Patients undergoing abdominal midline laparotomies(emergency or elective)
- Patients aged 18 years and above.

Exclusion criteria:

- Gynecological and urological surgeries.
- Those undergoing relaprotomy.
- Those known to be suffering from collagen vascular disease ,connective tissue disorders.
- Co-morbid factors like anemia, hypertension, diabetes mellitus, etc will be corrected where possible.
- Antibiotics to be started, as part of pre-operative treatment in all patients presenting with acute abdomen in emergency ward, and course will be prolonged accordingly in each case after operation.

- A prophylactic dose of antibiotics to be given in all elective cases along with extension of antibiotic as required.
- As a routine, in all cases the lineaalba to be closed with non-absorbable monofilament, synthetic suture (polypropylene No.1).
- Examination of wound will be started from **third post-operative day** onwards, and will include inspection for any redness, edema or presence of discharge like pus or serosangunious fluid.
- Examination to be continued till, suture removal and scar formation.

  All patients will be scored on basis of pre-operative ,operative and post-operative factors.

  A detailed clinical history will be taken for all the patients. Thorough physical examination will be done for all the patients.
- The calculation of the probability of abdominal wound dehiscence for an Individual surgical patient is performed in two steps-

1. First, the total risk score is calculated by adding the weights of the various variables as in Table 1.

RISK FACTORS	VARIABLES	RISK SCORE
PRE- OPERATIVE	AGE- 40-49 YRS 50-59 YRS 60-69 YRS >70 YRS	 0.4 0.9 0.9 1.1
	MALE	0.7
	CO-MORBID COPD	 0.7
	ASCITES	1.5
	JAUNDICE	0.5
	ANEMIA	0.7

RISK FACTORS	VARIABLE	RISK SCORE
OPERATIVE	EMERGENCY SURGERY	0.6
TYPE OF SURGERY-	GALL BLADDER/BILE DUCT	0.7
	ESOPHAGUS	1.5
	GASTRO-DUODENUM	1.4
	SMALL BOWEL	0.9
	LARGE BOWEL	1.4
	VASCULAR	1.3
POST-OPERATIVE	COUGHING	1.4
	WOUND INFECTION	0.9

2. In the second step, the probability of developing abdominal wound dehiscence, P, is calculated according to the logistic formula:

P= ex/ (1+ex)x100%; where 'ex' represents the exponential function and 'x' represents '-8.37 + (1.085 x calculated total risk score)'.

Then we compare this probability score with the actual outcome of the patient with regard to the occurrence of abdominal wound dehiscence.

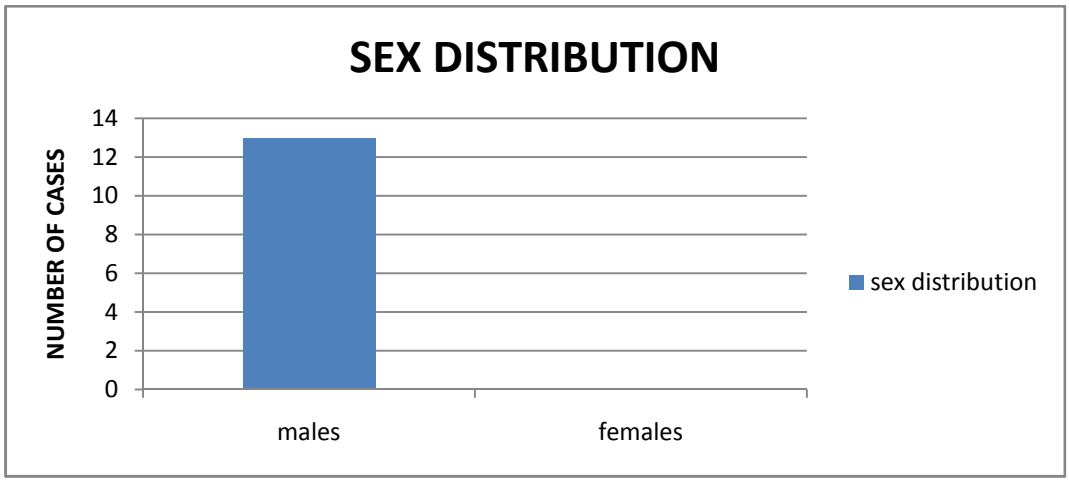
RESULTS

The risk model scoring system was applied to a total of 100 laparotomy cases approached by vertical midline incision comprising 76 male and 24 female patients including both elective and emergency abdominal surgeries. Out of the study population 13 patients developed abdominal wound dehiscence. All 13 cases were male patients.

SEX DISTRIBUTION-

Sex distribution	Male	Female	Total cases
No. of laparotomies	**76**	**24**	**100**
No. of abdominal wound dehiscence cases	**13**	**Nil**	**13**

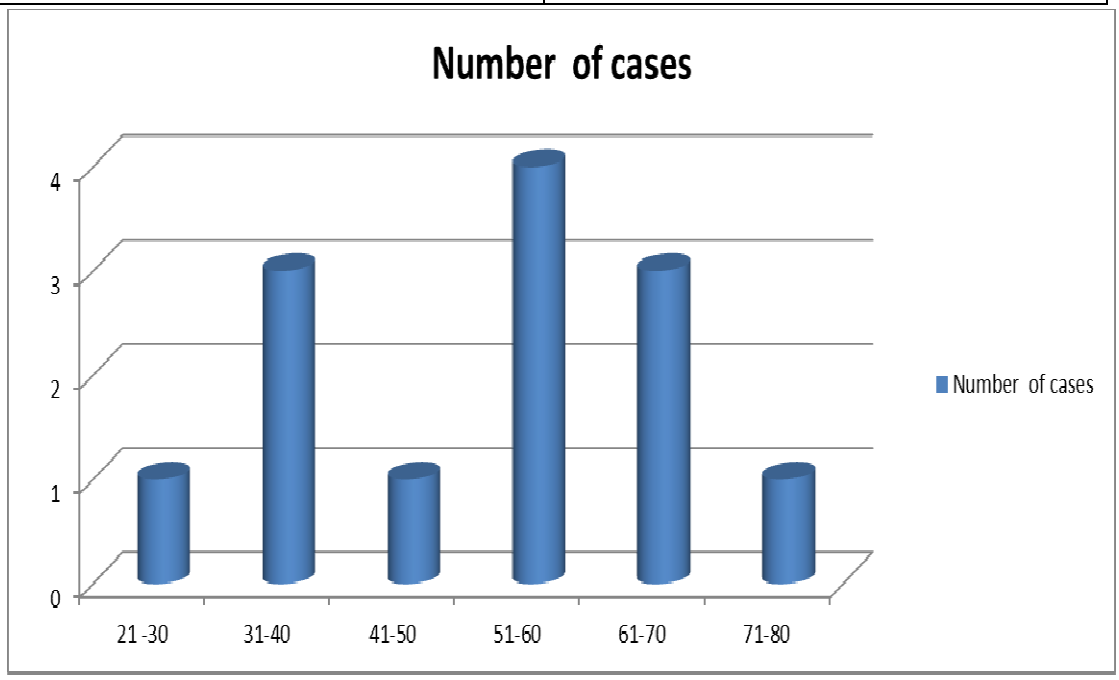
The occurrence of abdominal dehiscence was mainly seen among **males.**



2. INCIDENCE OF ABDOMINAL WOUND DEHISCENCE IN DIFFERENT AGE GROUPS-

Our study population consisted of patients between the ages ranging from 18yrs to 83 yrs. Majority of the patients who had abdominal wound dehiscence belonged to the age group 51 to 60 years. Youngest patient was 21 years old and the oldest patient affected was 72 years old.

Different age groups	Number of cases
21 – 30 years	1
31 -40 years	3
41-50 years	1
51 -60 years	4
61 -70 years	3
71 -80 years	1
Total	**13**



3. PREVALANCE OF CO-MORBID FACTORS AT THE TIME OF ADMISSION

Co-morbid factors at the time of admission	Number of cases	Percentage
Chronic obstructive pulmonary disease(COPD)	**6**	**46%**
Ascites	**3**	**23%**
Jaundice	**5**	**38%**
Anemia	**6**	**46%**

C.O.P.D and anemia were the major co-morbid factors associated with abdominal wound dehiscence.

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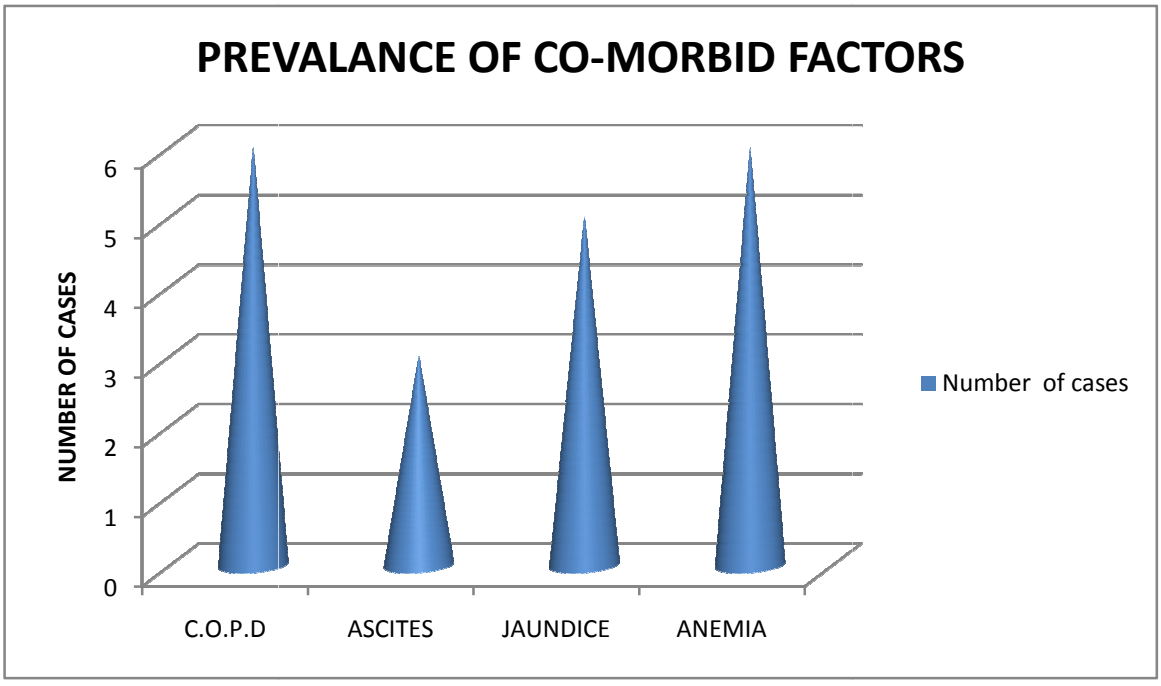
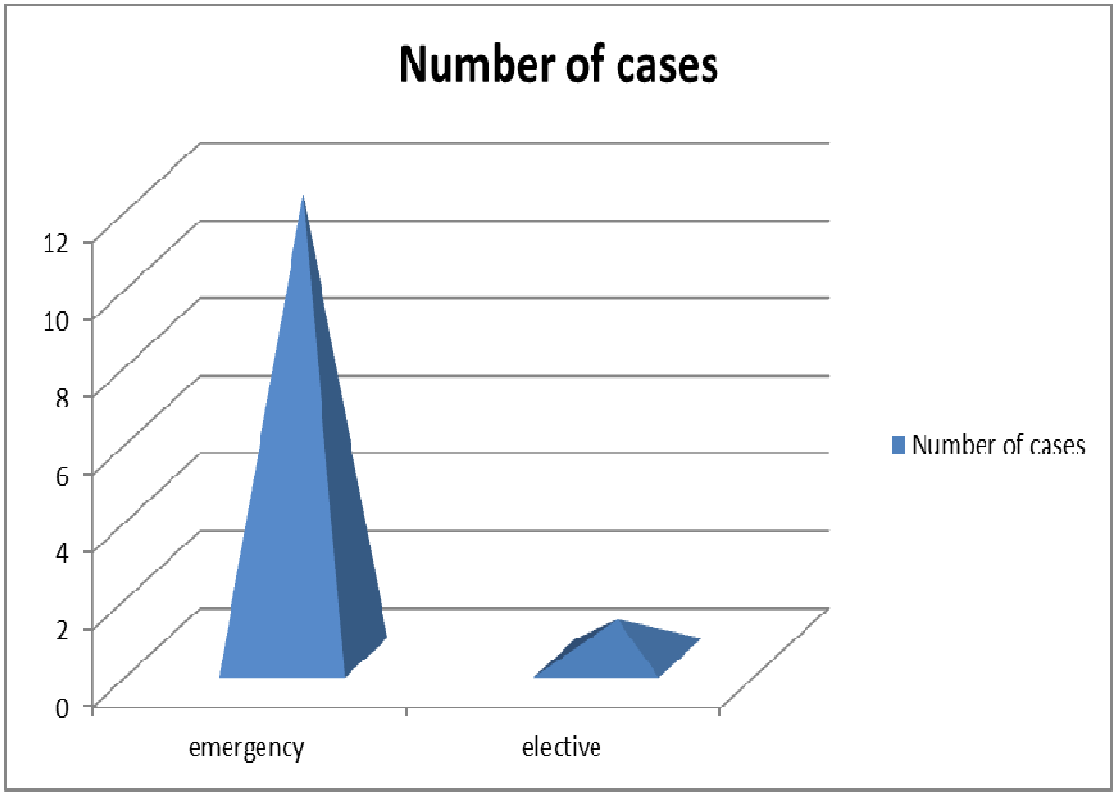


4. **EFFECT OF EMERGENCY SURGERY IN DEVELOPMENT OF ABDOMINAL WOUND DEHISCENCE-**

In our study population out of 100 patients, comprising 88 emergency and 12 elective cases. Total out of 13 cases of abdominal wound dehiscence 12 were emergency cases and the remaining one was an elective case.

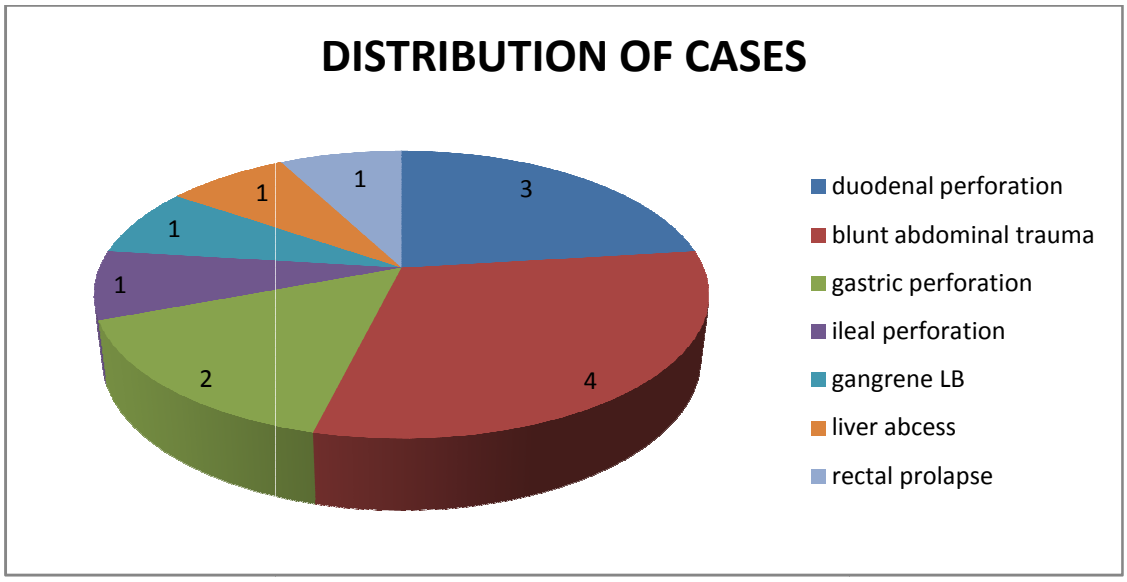
		No. of cases	Percentage
Surgery	Emergency	12	93%
	Elective	1	7%



5. DISTRIBUTION OF PATIENTS WITH ABDOMINAL WOUND DEHISCENCE ACCORDING TO INTRA-ABDOMINAL PATHOLOGY.

Out of 13 patients who had abdominal wound dehiscence, 10 patients comprised of hollow viscus perforation majority due to duodenal perforation and those secondary to blunt abdominal trauma. Blunt abdominal trauma contributed to 3 cases of ileal perforation and a case of jejunal perforation. Spontaneous ileal perforation secondary to abdominal TB with immune-compromised status was also noted. The rest comprised a case of large bowel gangrene, ruptured liver abscess and rectal prolapse.

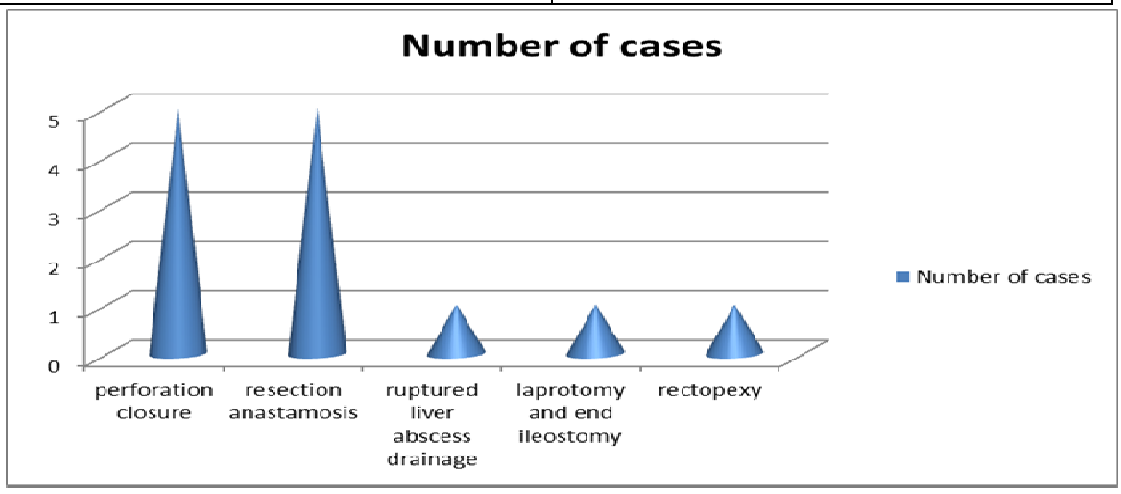
DISTRIBUTION OF CASES WITH ABDOMINAL WOUND DEHISCENCE ACCORDING TO INTRA-ABDOMINAL PATHOLOGY.



6. VARIOUS ABDOMINAL OPERATIVE PROCEDURES ENCOUNTERED WITH ABDOMINAL WOUND DEHISCENCE.

Out of 13 patients of abdominal wound dehiscence, perforation closure with live omental patch was performed in 5 cases, resection and anastomosis of small bowel done in 5 cases, and following cases of laparotomy and end ileostomy, laparotomy and drainage of liver abscess and rectopexy.

Operative procedure	Number of cases
Perforation closure	**5**
Resection and anastomosis	**5**
Laparotomy and drainage of liver abscess	**1**
Laparotomy and end ileostomy	**1**
Rectopexy	**1**
TOTAL	**13**



7. PATTERN OF PRESENTATION IN ABDOMINAL WOUND DEHISCENCE CASES.

Three patterns of occurrence of abdominal wound dehiscence was noted in the present study-

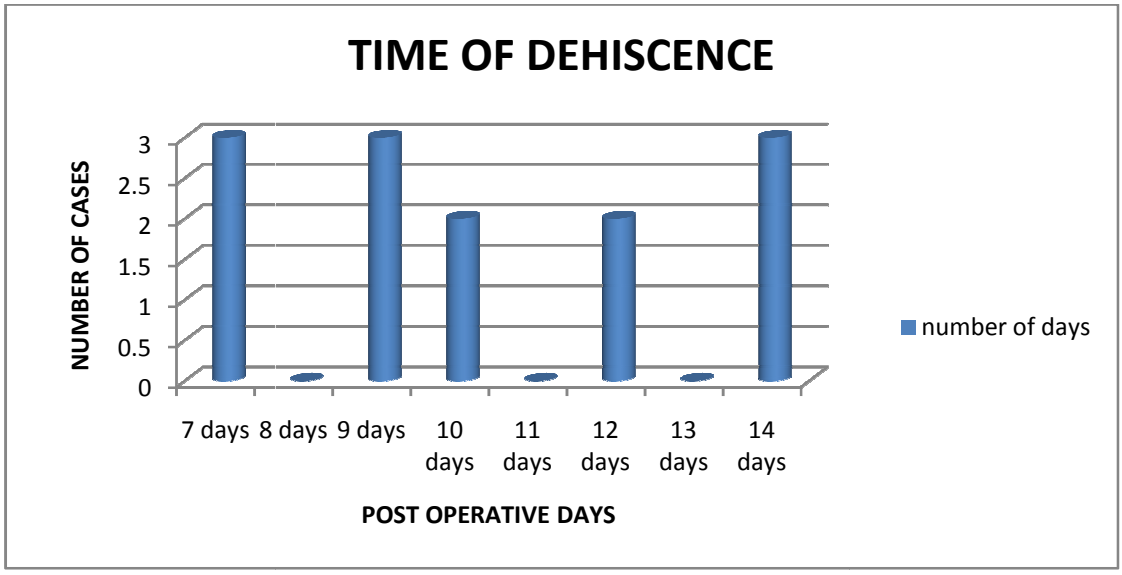
- Abrupt rush of pink serosanguineous discharge from the wound with a soft and boggy swelling noted underneath the skin following which wound exploration was done and confirmed dehiscence was noted in a majority of 7 cases.
- Sudden give away in the wound following excessive strain resulting in dehiscence was noted in 2 cases.
- Delayed onset of dehiscence in a setting of wound infection noted in 4 cases.

Types of presentation	Number of cases
Serousanguineous discharge with dehiscence underneath skin	**7**
Delayed onset of dehiscence with wound infection	**4**
Sudden give away in the wound	**2**

8. TIME OF DISRUPTION OF WOUND DEHISCENCE

Occurrence of burst abdomen in days range from 7 – 14 days with mean of wound dehiscence occurring at 10.3 days.

Out of total 13 cases which presented with abdominal wound dehiscence, there were equal number of cases seen occurring at 7^{th} POD, 9^{th} POD and 14^{th} POD.



9.POST-OPERATIVE FACTORS INFLUENCING THE OCCURRENCE OF ABDOMINAL WOUND DEHISCENCE.

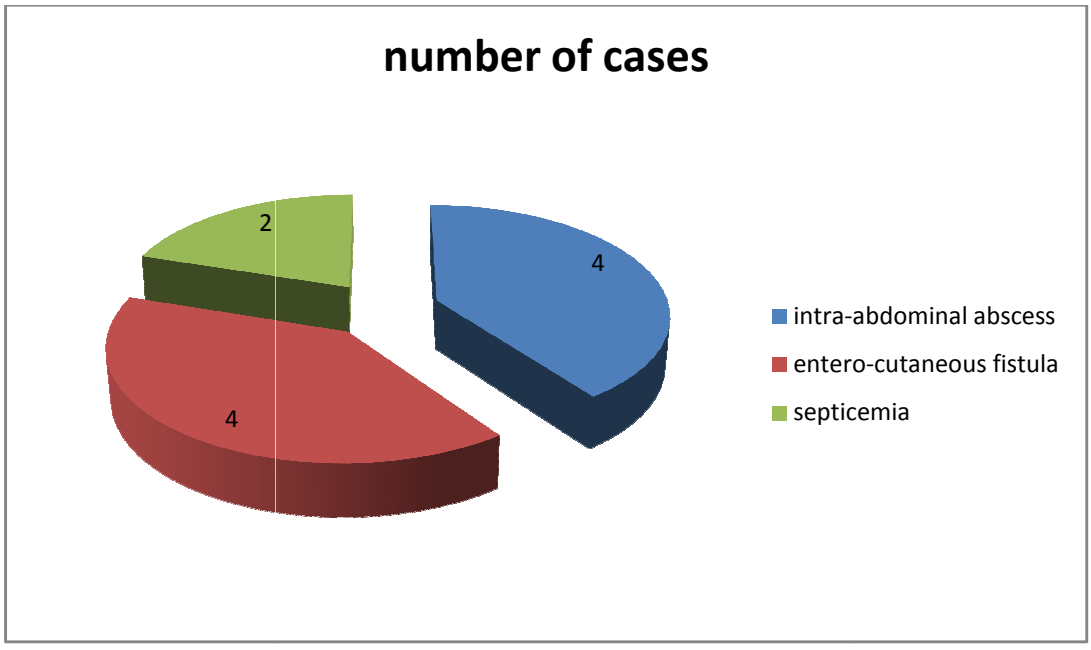
Post operative cough and wound infection were the two significant risk factors evaluated in the study in influencing the occurrence of wound dehiscence.

Out of 13 cases of abdominal wound dehiscence, 7 cases had significant post operative cough and 11 cases had wound infection.

RISK VARIABLE	POSITIVE	TOTAL
POST OPERATIVE COUGH	**7(53%)**	**13**
WOUND INFECTION	**11(84.6%)**	**13**

10. COMPLICATIONS ASSOCIATED WITH WOUND DEHISCENCE

Out of 13 cases of abdominal wound dehiscence, 4 patients developed intra-abdominal abscess and enterocutaneous fistula, 2 patients developed septicemia post operatively.



9.DURATION OF HOSPITAL STAY-

Range of hospital stay for all abdominal wound dehiscence cases was 18 -30 days.

Average hospital stay	**20 days.**

Hospital stay for more than 20 days was noted in 6 cases and were mainly due development of complications and co-morbid factors associated with the disease.

10.MORTALITY-

There was one death among the cases of wound dehiscence mainly due to development of septicemia and respiratory tract illness.

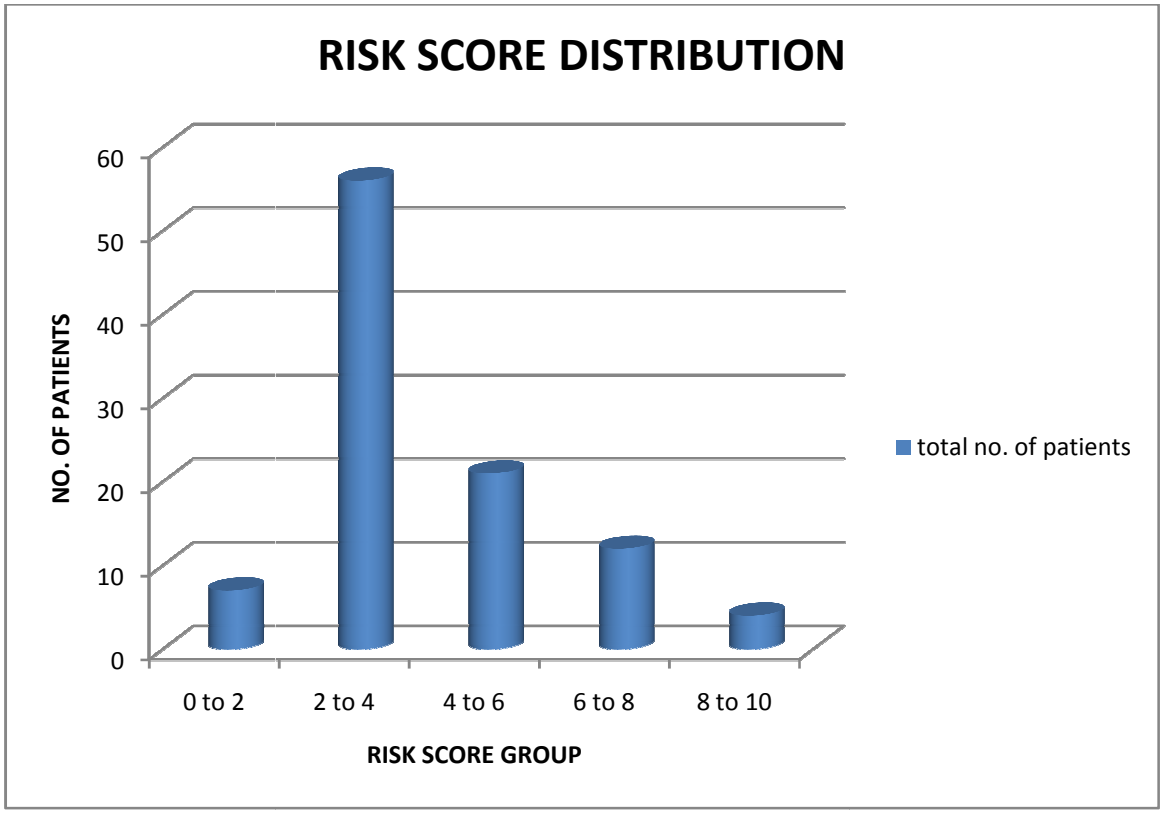
Total cases	**Survivals**	**Death**
13	**12(93%)**	**1(7%)**

11.MANAGEMENT OF WOUND DEHISCENCE-

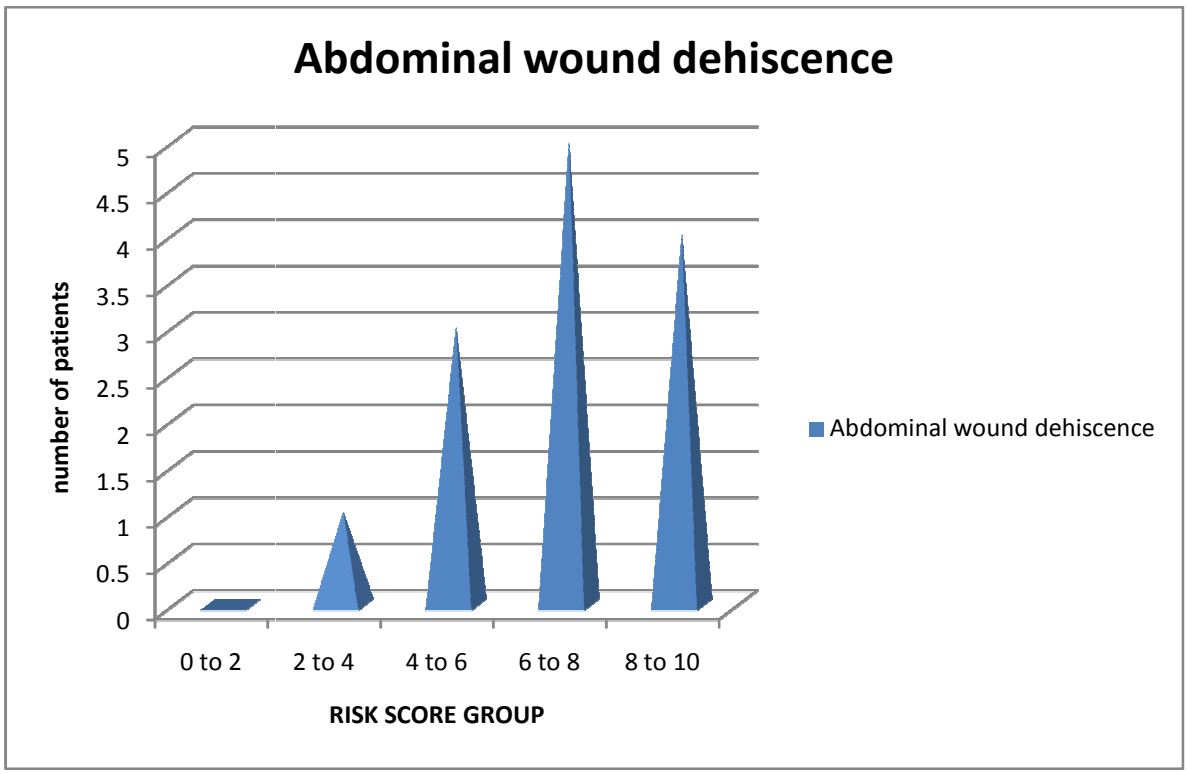
Types of wound dehiscence	Number of Patients	Management
Partial wound dehiscence	**10**	Conservative management (healing by secondary intention).
	6	Secondary suturing
Complete wound dehiscence	12	Retention sutures(tension suturing)

12.ABDOMINAL WOUND DEHISCENCE RISK MODEL IN STUDY POPULATION

Out of total 100 patients , majority belonged to the risk score group 2 – 4 which included 56 patients.



Distribution of patients with abdominal wound dehiscence according to risk score was noted as follows-



Out of 13 patients with abdominal wound dehiscence , Majority of patients who had abdominal wound dehiscence belonged to the risk group category 6 – 8 which included 5 patients.

The mean probability of developing wound dehiscence (percentage) was calculated in each risk score group and listed below-

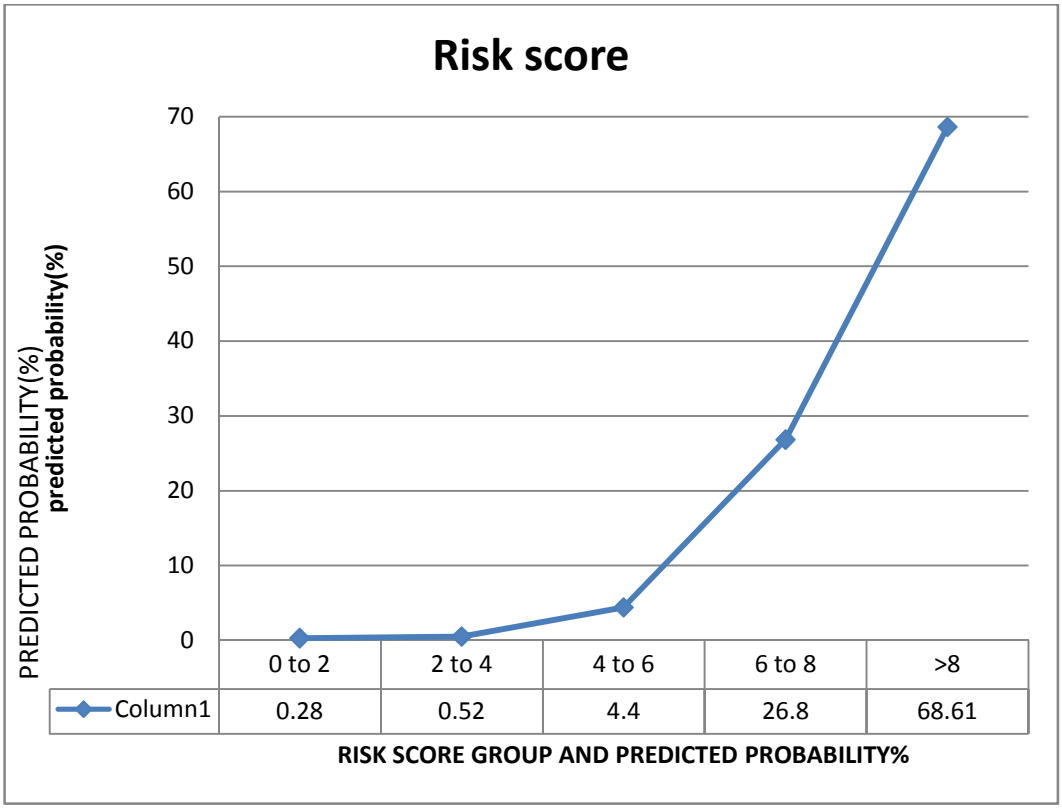
RISK SCORE	TOTAL NUMBER OF PATIENTS	ABDOMINAL WOUND DEHISCENCE		MEAN PROBABILITY (%)a
		NUMBER	PERCENT	
0 – 1.99	7	0	0.0	0
2 – 3.99	56	1	1.78%	0.52
4 -5.99	21	3	14.28%	4.48
6 – 7.99	12	5	41.6%	26.80
>8	4	4	100%	68.61
TOTAL	100	13	13%	

a. mean values of individual calculated probabilities according to risk score within risk score group.

The probability of developing wound dehiscence increases with the risk score in each group. The probability of developing wound dehiscence rises exponentially when the risk score gradually increases from 6 onwards. Patients in the risk score group 4 -6 had a mean probability of dehiscence 4.4% which there afterwards rises

exponentially to 26.8% and 68.6% in the risk scores group 6 -8 and >8 respectively. All patients with Risk scores >8 developed wound dehiscence with a mean probability of 68.61% chance of developing wound dehiscence.

Graph showing predicted probability (%) of developing abdominal wound dehiscence according to risk score.



Comparison between the predicted probability and actual percentage of abdominal wound dehiscence in study population-

RISK SCORE GROUP	PREDICTED PROBABILITY	ACTUAL PROBABILITY
0 – 1.99	0.28%	0
2 - 3.99	0.52%	1.78%
4 - 5.99	4.48%	14.28%
6 – 7.99	26.8%	41.6%
>8	68.61%	100%

The predicted probability of developing wound dehiscence as calculated according to the risk model nearly correlates or slightly under estimates when compared with the actual probability of wound dehiscence in the respective risk score groups, though it clearly shows an exponential rise in the probability of developing wound dehiscence with rise in risk score.

DISCUSSION

Burst abdomen represents a grave post-operative complication occurring despite advances in pre-operative and operative care in 21st century which carries with it a significant post- operative morbidity and mortality.

In our study population of 100 patients who underwent major abdominal midline laparotomies, 13 patients developed abdominal wound dehiscence which is comparatively higher than those mentioned in western literature.. The incidence rate in Wolff's study (1950) was 2.6% , Mann (1962) found 2.72% , Efron (1965) 2.2% , Lehman et al. (1968) 2.5% . Incidence of abdominal wound dehiscence in India was noted to be 5.6% in a study conducted at a hospital in Baroda.

Males were predominantly affected comprising of all the affected cases of abdominal wound dehiscence due the fact of smoking, alcohol consumption and chronic respiratory disorders more common in them.

It was found that there was increase in incidence of wound dehiscence among 51 – 60 yrs. age group of patients which correlates nearly with that mentioned in the literature. According to Maingot(Textbook of abdominal operations), average age is 45 years and that mentioned in Wolff study is it is four times more common in patients above 45 years than younger age group.

According to our study, pre-operative predisposing factors like chronic obstructive pulmonary disease and anemia were the major comorbid factors associated with wound dehiscence. In many patients there were more than one factor contributing to the occurrence of wound dehiscence. Joergerson, smith and Wolff noticed in their study a higher incidence of burst abdomen in patients with anemia, hypoproteinemia and vitamin deficiency. Hampton found out a higher incidence of burst abdomen in patients having pre-operative cough and chronic respiratory diseases which was found significant in our study as well.

In the present scoring system ,comorbid factors which were not studied were diabetes, malignancy, sepsis, post- operative vomiting, uremia, corticosteroid abuse as their effect was not considered significant in the present study, though they have been identified as risk factors by several authors.

The study is limited in evaluating additional risk factors like nutritional state which has a significant effect on wound healing.

It was found that emergency cases had a higher predisposition of developing wound dehiscence than elective ones occurring in 12 emergency cases out of 13 cases. according to afzal et al incidence in emergency laparotomy was 12.45% compared to elective one of

1.73%..Indian authors have reported burst abdomen to occur in 10 to 30% of emergency cases.

Hollow viscus perforation was the most common intra-abdominal pathology associated with wound dehiscence more commonly duodenal perforation and those due to blunt abdominal trauma. In most of the hollow viscus perforation ,peritoneal spillage of gastrointestinal tract contents lead to various forms of peritonitis and wound infection resulting in poor wound healing and dehiscence.

Fascial dehiscence after trauma laparotomy is associated with intra-abdominal sepsis, wound sepsis thus fascial dehiscence should be viewed as a sign of possible underlying intra-abdominal abscess.(24)

Among the operative procedures performed, perforation closure and resection anastomosis were the major procedure associated with abdominal wound dehiscence due to the fact of higher chance of anastomosis leak, intra-abdominal abscess formation leading to wound dehiscence.

Most common pattern of presentation of wound dehiscence was noticing a pink serosanguinous discharge from the wound with a soft and boggy swelling underneath the skin which correlates with the study mentioned in Maingot.(Text book of abdominal operations).

Average time of occurrence of abdominal wound dehiscence was at 9th POD. According to a study at the long island Jewish medical center ,during a 5 year period from jan1984 to jan 1989, serosanguinous discharge was noted from the wound prior to dehiscence in 21 patients(67%) and disruption occurred on an average of 11.1 post-operative days which correlates well with our study.

Out of 13 cases of burst abdomen , 7 patients had post-operative cough(53%), 11 cases had associated wound sepsis(84%), thus proving wound infection to be a major determinant in occurrence of burst abdomen .30% of burst abdomen cases were reported in wound infected cases by professor Naithani's unit from Allahabad. According to the traditional wound classification, wound infections occur in-

- 3.6% of clean wounds.
- 8.4% of clean –contaminated wounds
- 11.8% of contaminated wounds.
- 31% of dirty wounds.

In several literature wound infection has been proved as the major determinant in causation of burst abdomen(25). Wound infection continues to be the major source of mortality and morbidity post-operatively.

Most common complication associated with burst abdomen were intra-abdominal abscess and enterocutaneous fistula. According toTillou et al fascial dehiscence should be viewed as a possible sign of possible underlying intra-abdominal abscess. Thus imaging or direct visualization of entire abdominal cavity is mandatory before management of dehisced fascia.

Average duration of hospital stay was 20 days. Hospital stay more than 20 days was noted in 6 cases of abdominal wound dehiscence mainly due to development of complications and associated comorbid factors with the disease.

Mortality following burst abdomen was noted in one case operated for large bowel gangrene which occurred following development of septicemia and respiratory complications. Mortality over the years has been steadily decreasing with Wolff reporting mortality as low as 11%. Management of complete wound dehiscence which occurred in a total of 12 patients was managed by retention sutures immediately after the occurrence of dehiscence. Partial wound dehiscence occurred in a total of 16 patients, were only a part of the wound was given away without evisceration occurred in 6 patients which was managed conservatively by secondary suturing after the control of wound infection and in patients were separation of only the superficial layers of abdomen which

occurred in 10 patients were managed conservatively by repeated daily dressings and allowing them to heal by secondary intention.

Very few studies have been done to predict the occurrence of abdominal wound dehiscence. Webster risk index which was developed in veteran affair population to stratify the risk according to scoring system was criticized for the lack of validation.

In our present study out of 13 patients who developed abdominal wound dehiscence, majority of patients belonged to the risk group 6 -8 which included 5 patients. There was a steady increase in the incidence of wound dehiscence with increase in risk score.

Mean probability of developing wound dehiscence was calculated in each risk score group which showed an exponential increase in the probability with the rise in risk score, which is comparable to the study done at the department of general surgery, Erasmus university medical center, Netherlands.(vanramshorst et al).

The mean probability of occurrence of dehiscence was compared with the actual occurrence of abdominal wound dehiscence which nearly correlated or slightly underestimates the actual occurrence even though the exponential rise in probability of abdominal wound dehiscence was noted.

RISK SCORE GROUP	PREDICTED PROBABILITY	ACTUAL PROBABILITY
0 – 1.99	0.28%	0
2 - 3.99	0.52%	1.78%
4 - 5.99	4.48%	14.28%
6 – 7.99	26.8%	41.6%
>8	68.61%	100%

Risk scores greater than 6 showed probability of 26% with nearly half of the patients actually developing wound dehiscence in this category and with risk scores greater than 8 all patients developed wound dehiscence with a mean probability of 68% showing high rates of dehiscence.

Thus we can stratify patients into low risk and high risk according to the risk score.

RISK SCORE	CATEGORY	PREDICTED PROBABILITY	NUMBER OF PATIENTS
<6	**Low risk**	**4.48%**	**4**
>6	**High risk**	**26.8%**	**9**

In our study out of 13 cases of abdominal wound dehiscence-

- 9 cases belonged to the high risk category with occurrence of 70% among the dehiscence cases.
- 4 cases belonged to low risk category with occurrence of 30% among the dehiscence cases.

Based on this classification we can triage the patients in the government hospitals where the patients are in plenty but the resources are limited.

CONCLUSION

Out of the abdominal wound dehiscence cases-

- Majority of the patients belonged to the age group of 5^{th} to 6^{th} decade.
- Male predominance in affection of abdominal wound dehiscence occurring in all 13 cases.
- Chronic obstructive pulmonary disease and anemia were the major comorbid preoperative factors associated with abdominal wound dehiscence.
- Wound infection being the single most important determinant in occurring of abdominal wound dehiscence occurring in 85% of cases.
- Nearly all cases occurred in a setting of emergency procedure.
- Death following occurrence of wound dehiscence was noted in one case as a result of septicemia and development of respiratory complications.
- All cases of complete wound dehiscence were managed immediately by tension suturing and partial wound dehiscence cases were managed according to the depth of dehiscence and presence of wound infection either conservatively or secondary suturing.
- Though the predicted probability calculated by the abdominal risk model may underestimate the actual occurrence of wound dehiscence,

there is a noticeable exponential increase in occurrence of abdominal wound dehiscence with increase in risk score.

Burst abdomen is a serious sequel to impaired wound healing. Knowledge of the common risk factors and how to treat these hazards should help to reduce the incidence of this dangerous complication. Proper and early diagnosis of burst abdomen and effective treatment decreases the morbidity and mortality of this complication.

We conclude that the abdominal risk model is a very good tool for predicting the occurrence of abdominal wound dehiscence and helps to classify and triage the patients to different groups , in whom preventive strategies can be instituted and thus reducing the risk of its occurrence.

Appendix-1

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Appendix 2

PROFORMA

Patient particulars

- Name
- Age
- Sex
- IP No
- Address
- DOA
- DOS
- DOD

History

- Complaints
- History of present illness
- History of Chronic Diseases

General Physical Examination

- Pulse
- Blood Pressure
- Temperature
- Hydration
- GCS

Examination of Abdomen

- Inspection
- Palpation
- Percussion
- Auscultation
- PR examination

Systemic Examination

- Respiratory System
- Cardiovascular System
- Central Nervous System

Investigations

- Hemoglobin
- blood sugar
- serum creatinine
- Blood Urea
- Serum Bilirubin
- Chest X-Ray
- Erect X-Ray Abdomen
- USG Abdomen

Operative Notes

- Diagnosis
- procedure
- Anesthesia

Post Operative Period

- Wound Infection
- Wound Dehiscence
- Respiratory Complications
- enterocutaneous fistula
- Intra Abdominal Abscess

MASTER CHART
Appendix -3

serial no.	NAME	AGE/SEX	IP NO.	D.O.A	D.O.D	COPD	ASCITES	JAUNDICE	ANAEMIA	ADMISSION	ORGAN	POST OP COUGH	WOUND INFECTION	SCORE	PRED.PROBABILITY	WOUND DEHISCENCE	TIME OF DISRUPTION
1	DHANAPATHY	20/M	66570	2.11.12	12.11.12	NEG	NEG	NEG	NEG	EMER	DUP	NEG	NEG	2.7	0.43%	NEG	NA
2	RAJENDRA	35/M	65403	25.10.13	2.11.13	NEG	NEG	NEG	NEG	EMER	DUP	NEG	NEG	2.7	0.43%	NEG	NA
3	RAJ KUMAR	23/M	59345	30.9.13	10.10.13	NEG	NEG	NEG	NEG	EMER	DUP	NEG	NEG	2.7	0.43%	NEG	NA
4	PANEER S	23/M	51658	26.8.13	6.9.13	NEG	NEG	NEG	NEG	EMER	GASP	NEG	NEG	2.7	0.43%	NEG	NA
5	HENSANY	21/M	29433	19.5.13	1.6.13	NEG	NEG	NEG	NEG	EMER	DUP	NEG	NEG	2.7	0.43%	NEG	NA
6	SIVAKUMAR	18/M	21988	15.4.13	10 5 13	NEG	NEG	NEG	NEG	EMER	ILEUM GANG	NEG	POS	4.1	1.94%	NEG	NA
7	VELUSAMY	72/M	20424	8.4.13	25.4.13	POS	NEG	NEG	POS	EMER	DUP	POS	NEG	6.60	22.99%	**POS**	**9TH POD**
8	RAJENDRAN	35/M	65320	2.2.13	13.2.13	NEG	NEG	NEG	POS	EMER	TRAUMA-GAS	NEG	POS	5.3	6.78%	**NEG**	NA
9	SAMPATH	19/M	61919	8.8.12	20.8.13	NEG	NEG	NEG	NEG	EMER	DUP	NEG	POS	4.6	3.30%	NEG	NA
10	PONNUSAMY	62/M	61899	12.8.12	1.9.12	POS	POS	POS	POS	EMER	LIVER ABC	POS	POS	8.9	78.35%	**POS**	**7TH POD**
11	NANJAPPAN	50/M	61901	20.8.12	30.8.12	NEG	NEG	NEG	POS	ELEC	DUU	NEG	NEG	3.7	1.26%	NEG	NA
12	PERIASAMY	42/M	62909	27.8.12	20.9.12	POS	NEG	NEG	NEG	EMER	SIG VOLV	POS	NEG	5.2	6.13%	NEG	NA
13	RABHABDULLA	48/M	67434	23.9.12	##########	NEG	POS	NEG	NEG	EMER	LB GANG	NEG	POS	6.5	21.12%	**POS**	**14TH POD**
14	CHINNAN	70/M	64543	30.9.12	11.10.12	NEG	POS	NEG	NEG	EMER	DUP	NEG	POS	7.2	36.40%	**NEG**	NA
15	MANI	40/M	67871	21.10.12	2.11.12	NEG	NEG	NEG	NEG	EMER	BLADDER	NEG	NEG	1.7	0.14%	NEG	NA
16	MALIK	21/M	24386	1.8.12	1.9.12	NEG	POS	POS	NEG	EMER	TRAUMA-SB	POS	POS	7.4	41.55%	**POS**	**7TH POD**
17	NATARAJ	65/M	18002	31.8.12	21.9.12	NEG	NEG	POS	NEG	EMER	GASP	NEG	POS	6	13.47%	**POS**	**10TH POD**
18	GOVINDA	53/M	60833	25.10.12	16.11.12	NEG	NEG	POS	NEG	EMER	TRAUMA-SB	NEG	POS	5.5	8.30%	**POS**	**10TH POD**
19	MURUGESHAN	39/M	43415	27.7.12	17.8.12	NEG	NEG	POS	NEG	EMER	ILEUM PERF	NEG	POS	4.6	3.30%	**POS**	**9TH POD**
20	DEVARAJ	45/M	61805	21.8.13	15.9.13	NEG	NEG	NEG	pos	EMER	SB OBS	NEG	POS	5.1	5.53%	**NEG**	NA
21	MANOJ	65/M	61123	12.8.13	30.8.13	POS	NEG	NEG	POS	EMER	SB OBS	NEG	POS	6.4	19.37%	**NEG**	NA
22	MUTUSAMY	65/M	67886	21.8.13	10.9.13	POS	NEG	NEG	POS	EMER	SB OBS	NEG	POS	6.4	19.37%	NEG	NA
23	DHANDAPANI	55/M	34806	11.6.13	24.6.13	NEG	NEG	NEG	POS	EMER	GASP	NEG	NEG	4.3	2.40%	NEG	NA
24	MURUGAN	45/M	43560	21.6.13	2.7.13	NEG	NEG	NEG	NEG	EMER	DUP	NEG	NEG	3.1	0.66%	NEG	NA
25	THANGAVEL	51/M	36909	26.6.13	15.7.13	POS	NEG	NEG	POS	EMER	TRAUMA-SB	POS	POS	7.8	52.32%	**POS**	**12TH POD**
26	PALANI GOVINDU	83/M	370301	21.1.13	2.2.13	POS	NEG	NEG	NEG	EMER	LB OBS	NEG	NEG	4.5	2.97%	NEG	NA
27	AMEER BASHA	47/M	38414	8.6.13	18.6.13	NEG	NEG	NEG	NEG	EMER	DUP	NEG	NEG	3.1	0.66%	NEG	NA
28	SARVANAN	47/M	38547	10.6.13	20.6.13	NEG	NEG	NEG	NEG	EMER	DUP	NEG	NEG	3.1	0.66%	NEG	NA
29	RAVI	28/M	42551	16.7.13	26.7.13	NEG	NEG	NEG	NEG	EMER	GASP	NEG	NEG	2.7	0.43%	NEG	NA
30	AYAPPAN	45/M	43291	20.7.13	30.7.13	NEG	NEG	NEG	NEG	EMER	SB GANG	NEG	NEG	2.6	0.38%	NEG	NA
31	SIVASUBRAMANI	42/M	44336	24.7.13	29.7.13	NEG	NEG	NEG	NEG	EMER	GASP	NEG	NEG	3.1	0.66%	NEG	NA
32	ASADUL	35/M	44347	24.7.13	5.8.13	NEG	NEG	NEG	NEG	EMER	DUP	NEG	NEG	2.7	0.43%	NEG	NA
33	PATCHIAMMAL	60/F	45495	30.7.13	10.8.13	NEG	NEG	NEG	NEG	EMER	DUP	NEG	NEG	2.9	0.53%	NEG	NA
34	SIVAPERASTHA	20/M	43236	29.7.13	8.8.13	NEG	NEG	NEG	NEG	EMER	DUP	NEG	NEG	2.7	0.43%	NEG	NA
35	DURAIKANNAN	50/M	46932	5.8.13	20.8.13	POS	NEG	NEG	NEG	EMER	ILEUM PERF	NEG	NEG	3.2	0.74%	NEG	NA
36	BABU	47/M	47220	6.8.13	17.8.13	NEG	NEG	NEG	NEG	EMER	SB OBS	NEG	NEG	2.6	0.38%	NEG	NA
37	NATCHAMMAL	65/F	904	5.1.13	16.1.13	NEG	NEG	NEG	POS	EMER	DUP	NEG	NEG	3.6	1.13%	NEG	NA
38	MANIYAMMAL	65/F	24671	3.8.12	22.8.12	NEG	NEG	NEG	POS	EMER	LB OBS	NEG	NEG	3.6	1.13%	NEG	NA
39	LAKSHMI	55/F	26265	10.5.13	26.5.13	NEG	NEG	NEG	NEG	EMER	SB OBS	NEG	NEG	2.4	0.31%	NEG	NA
40	VALLIYAMMAL	50/F	17072	20.12.12	29.12.12	NEG	NEG	NEG	POS	ELEC	GAS CA	NEG	NEG	3	0.60%	NEG	NA
41	TAMILARASI	60/F	503	22.1.13	30.1.13	NEG	NEG	NEG	POS	ELEC	GAS CA	NEG	NEG	3	0.60%	NEG	NA
42	IYYAMMAL	40/F	11873	28.2.13	10.3.13	NEG	NEG	NEG	POS	ELEC	GAS CA	NEG	NEG	2.5	0.34%	NEG	NA
43	RUCKMANI	50/F	36791	20.6.13	1.7.13	NEG	NEG	NEG	POS	ELEC	LB OBS	NEG	NEG	3	0.60%	NEG	NA
44	KALYANI	80/F	44491	21.7.13	2.8.13	NEG	NEG	NEG	POS	EMER	GASP	NEG	POS	5.7	10.10%	NEG	NA
45	SHILPA	18/F	62208	13.10.13	20.10.13	NEG	NEG	NEG	NEG	EMER	APP PERF	NEG	NEG	1.5	0.11%	NEG	NA

46	VELUMANI	45/F	53927	6.9.13	22.9.13	NEG	NEG	POS	NEG	ELEC	CBD STRICTU	NEG	NEG	1.6	0.30%	NEG	NA
47	ACHIKANNU	60/F	28872	17.5.13	25.5.13	NEG	NEG	NEG	POS	ELEC	GAS CA	NEG	NEG	3	0.60%	NEG	NA
48	RAMAYEE	75/F	52506	16.8.13	25.8.13	NEG	NEG	NEG	POS	EMER	LB OBS	NEG	NEG	3.8	1.41%	NEG	NA
49	CHINAMMMAL	80/F	51398	4.8.12	19.8.12	NEG	NEG	NEG	POS	ELEC	LB OBS	NEG	NEG	3.2	0.74%	NEG	NA
50	MARIAMMAL	42/F	58030	1.10.13	10.10.13	NEG	NEG	NEG	NEG	EMER	SB OBS	NEG	NEG	1.9	0.18%	NEG	NA
51	KANDIYAMMAL	41/F	59669	1.10.13	7.10.13	NEG	NEG	NEG	NEG	EMER	APP PERF	NEG	NEG	1.9	0.18%	NEG	NA
52	PACHIYAMMAL	60/F	45495	30.7.13	10.8.13	NEG	NEG	NEG	POS	EMER	DUP	NEG	NEG	3.6	1.13%	NEG	NA
53	KANAGA L	73/F	2364	13.1.13	26.1.13	NEG	NEG	NEG	POS	EMER	APP PERF	NEG	NEG	3.3	0.82%	NEG	NA
54	LAKSHMI	80/F	12415	8.8.12	21.8.12	NEG	NEG	NEG	NEG	EMER	SB OBS	NEG	NEG	2.6	0.38%	NEG	NA
55	RAMATHAL	70/F	65969	20.11.12	1.12.12	NEG	NEG	NEG	NEG	EMER	SB OBS	NEG	NEG	2.6	0.38%	NEG	NA
56	MANONMANI	30/F	47471	22.8.12	1.9.12	NEG	NEG	NEG	NEG	EMER	APP PERF	NEG	NEG	1.5	0.11%	NEG	NA
57	SAROJINI	22/F	47518	22.8.12	2.9.12	NEG	NEG	NEG	NEG	EMER	APP PERF	NEG	NEG	1.5	0.11%	NEG	NA
58	POOVAL	45/F	43398	20.7.13	2.8.13	NEG	NEG	NEG	POS	EMER	LB OBS	NEG	NEG	3.1	0.66%	NEG	NA
59	PALISHWARI	25/F	13482	10.8.12	22.8.12	NEG	NEG	NEG	POS	EMER	SB OBS	NEG	NEG	2.2	0.25%	NEG	NA
60	KUMAR	60/M	64625	23.10.13	10.11.13	NEG	NEG	NEG	POS	EMER	DUP	NEG	POS	6.2	16.20%	**NEG**	NA
61	SUBRAMANI	67/M	58342	27.9.13	15.10.13	POS	NEG	NEG	POS	EMER	DUP	POS	POS	8.3	65.37%	**POS**	**12TH POD**
62	BHUVANESHWAR	18/M	52921	1.9.13	9.9.13	NEG	NEG	NEG	NEG	EMER	APP PERF	NEG	NEG	2.2	0.25%	NEG	NA
63	SHAHID A	18/M	37654	8.7.13	13.7.13	NEG	NEG	NEG	NEG	EMER	APP PERF	NEG	NEG	2.2	0.25%	NEG	NA
64	SARVANAN	37/M	47511	8.8.13	17.8.13	NEG	NEG	NEG	NEG	EMER	DUP	NEG	NEG	2.7	0.43%	NEG	NA
65	PALANISAMY	37/M	17530	25.7.13	5.8.13	NEG	NEG	NEG	POS	EMER	LB OBS	NEG	POS	5.3	6.78%	**NEG**	NA
66	KRISHNAN	45/M	43652	21.7.13	3.8.13	NEG	NEG	NEG	NEG	EMER	DUP	NEG	NEG	3.1	0.66%	NEG	NA
67	ENALAPPAN	33/M	43970	16.7.13	24.7.13	NEG	NEG	NEG	NEG	EMER	APP PERF	NEG	NEG	2.2	0.25%	NEG	NA
68	BALAMAHENDRAN	19/M	41435	11.7.13	17.7.13	NEG	NEG	NEG	NEG	EMER	APP PERF	NEG	NEG	2.2	0.25%	NEG	NA
69	THANGAVEL	50/M	36909	25.6.13	#######	NEG	NEG	NEG	NEG	EMER	TRAUMA-SB	NEG	POS	5	5%	NEG	NA
70	PARTHIBAN	18/M	36764	20.6.13	27.6.13	NEG	NEG	NEG	NEG	EMER	APP PERF	NEG	POS	4.1	1.94%	NEG	NA
71	MANTHRAPAN	75/M	28704	16.5.13	2.6.13	POS	NEG	NEG	POS	ELEC	SB OBS	NEG	POS	6	13.47%	**NEG**	NA
72	KARUMALAYA	35/M	28784	16.5.13	24.5.13	NEG	NEG	NEG	NEG	EMER	DUP	NEG	NEG	2.7	0.43%	NEG	NA
73	MURUGAN	45/M	30186	23.5.13	2.6.13	NEG	NEG	NEG	NEG	EMER	DUP	NEG	NEG	3.1	0.66%	NEG	NA
74	SALEEM	74/M	27242	9.5.13	18.5.13	NEG	NEG	NEG	POS	EMER	GAS CA	NEG	NEG	4.5	2.97%	NEG	NA
75	MURUGAN	45/M	24238	23.4.13	5.5.13	NEG	NEG	NEG	NEG	EMER	TRAUMA-SB	NEG	NEG	2.6	0.38%	NEG	NA
76	MARUTHACHLAM	51/M	24957	29.4.13	15.5.13	POS	NEG	NEG	POS	EMER	DUP	POS	POS	8.3	65.37%	**POS**	**14TH POD**
77	SIVA	19/M	19621	4.4.13	#######	NEG	NEG	NEG	POS	EMER	DUP	NEG	POS	5.3	6.78%	NEG	NA
78	SHAHID	24/M	14904	14.3.13	23.4.13	NEG	NEG	NEG	NEG	EMER	DUP	NEG	NEG	2.7	0.43%	NEG	NA
79	MOHD. MOSA	45/M	62052	11.1.13	20.1.13	NEG	NEG	NEG	POS	EMER	CA RECTUM	NEG	NEG	3.8	1.41%	NEG	NA
80	MANNAN	21/M	4563	25.1.13	3.2.13	NEG	NEG	NEG	NEG	EMER	DUP	NEG	NEG	2.7	0.43%	NEG	NA
81	AJITH	32/M	65938	10.1.13	19.1.13	NEG	NEG	NEG	NEG	EMER	GASP	NEG	NEG	2.7	0.43%	NEG	NA
82	MANU	36/M	61997	10.1.13	18.1.13	NEG	NEG	NEG	NEG	EMER	DUP	NEG	NEG	2.7	0.43%	NEG	NA
83	BABA	59/M	25103	29.4.13	5.5.13	NEG	NEG	NEG	NEG	EMER	APP PERF	NEG	POS	5	5%	NEG	NA
84	SURESH	40/M	71238	25.2.13	3.3.13	NEG	NEG	NEG	NEG	EMER	DUP	NEG	POS	5	5%	NEG	NA
85	RAJENDRAN	50/M	9752	18.2.13	27.8.13	NEG	NEG	NEG	NEG	EMER	DUP	NEG	NEG	3.6	1.13%	NEG	NA
86	NAGARAJ	45/M	5733	28.1.13	7.2.13	NEG	NEG	NEG	NEG	EMER	DUP	NEG	NEG	3.1	0.66%	NEG	NA
87	RAVI	45/M	2408	13.1.13	22.1.13	NEG	NEG	NEG	NEG	EMER	GASP	NEG	NEG	3.1	0.66%	NEG	NA
88	GOPAL K	40/M	50901	20.7.13	10.8.13	NEG	NEG	NEG	NEG	**ELEC**	RECAL PROLA	NEG	NEG	2.5	0.34%	**POS**	**7TH POD**
89	KAMALADAS	40/M	51406	25.8.13	14.9.13	NEG	NEG	NEG	NEG	EMER	TRAUMA-SB	NEG	POS	4.5	2.97%	**POS**	**9TH POD**
90	GEORGE	45/M	52234	9.1.13	21.1.13	NEG	NEG	NEG	NEG	EMER	TRAUMA-SB	NEG	POS	4.5	2.97%	**NEG**	NA
91	ARUNACHALAM	50/M	45565	4.8.12	28.8.12	POS	NEG	NEG	NEG	EMER	SB GANG	POS	POS	7.1	33.92%	**NEG**	NA
92	SOMASUNDARAM	55/M	60969	8.6.13	28.6.13	POS	NEG	NEG	POS	EMER	GASP	POS	POS	8.3	65.37%	**POS**	**14TH POD**
93	PALANISAMY	52/M	51230	10.6.13	20.6.13	NEG	POS	NEG	NEG	EMER	GASP	NEG	POS	7	31.53%	**NEG**	NA
94	SATHISH	29/M	66543	25.8.12	4.9.12	NEG	NEG	NEG	NEG	EMER	DUP	NEG	NEG	2.7	0.43%	NEG	NA
95	SELVARAJ	42/M	58947	28.9.13	8.10.13	NEG	NEG	NEG	POS	EMER	SB OBS	NEG	NEG	3.3	0.82%	NEG	NA
96	RAJU	60/M	54192	7.8.13	17.8.13	NEG	NEG	NEG	NEG	ELEC	GAS CA	NEG	NEG	3	0.59%	NEG	NA
97	THIRUMURTHI	60/M	32328	1.6.13	17.6.13	NEG	NEG	NEG	POS	ELEC	LB OBS	NEG	NEG	3.7	1.26%	NEG	NA
98	MANIKANDAN	25/M	9433	15.2.13	25.2.13	NEG	NEG	NEG	NEG	EMER	DUP	NEG	NEG	2.7	0.43%	NEG	NA
99	GANESAN	30/M	61189	26.1.13	5.1.13	NEG	NEG	NEG	NEG	EMER	GASP	NEG	NEG	2.7	0.43%	NEG	NA
100	SHANMUGAM	39/M	65055	27.10.13	3.11.13	NEG	NEG	NEG	NEG	EMER	APP PERF	NEG	POS	4.1	1.94%	**NEG**	NA

LEGENDS

DOA- Date of Admission;

DOD- Date of Discharge; ;

S. BILI- Serum Bilirubin;

COPD- chronic obstructive airway disease;

DUP- Duodenal of Perforation;

ILEUM PERF- ileum Perforation;

GASP- Gastric Perforation;

GAS CA- gastric carcinoma;

SB OBS- small bowel obstruction;

LB OBS- large bowel obstruction;

SB GANG –small bowel gangrene;

LB GANG- large bowel gangrene;

ILEUM GANG- ileum gangrene ;

TRAUMA SB- trauma small bowel;

ILEUM PERF- ileum perforation;

APP PERF- appendicular perforation;

CBD STRIC- cbd stricture ;

RECTUM PROLA- rectum prolapse;

POD- post operative day;

neg- negative; pos- positive;

NA- not applicable;

emer- Emergency Surgery;